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Paul R. Miller

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CONTENTS

SCREENING OF FUNGICIDES AND CHEMOTHERAPEUTANTS FOR CONTROL OF PINK ROOT OF ONIONS AND SHALLOTS[1]

M. M. Kulik and E. C. Tims[2]

Summary

The filter paper disc method of evaluating fungicides in the laboratory was modified to permit the use of Pyrenochaeta terrestris, the cause of pink root, as the test organism. Twenty-five out of 54 fungicides and 8 out of 29 chemotherapeutants tested at 1000 ppm by this method yielded highly significant results. In addition, two other fungicides yielded significant results.

Twenty-two out of 50 fungicides and none of the 29 chemotherapeutants tested on onions in sand culture at 1000 ppm reduced the incidence of pink root to 1% or less. Twenty of the 22 effective fungicides were later tested at 200 ppm and 9 of them reduced the disease incidence to 1% or less.

A number of field tests were set up over a 2-year period in attempts to correlate results of laboratory, greenhouse, and field experiments. The results were inconclusive because of lack of sufficient pink root development.

INTRODUCTION

Although pink root is one of the more serious diseases of onions and shallots in Louisiana no satisfactory practices for the control of the disease have been established. Since many new chemicals had become available and there were reports of successful trials with various fungicides (1, 3, 4, 5), a screening program was initiated to determine the relative efficiency of some of the new materials under laboratory and greenhouse conditions. Subsequently an attempt was made to correlate the results obtained with the action of the chemicals under field conditions.

LABORATORY SCREENING

The agar plate method commonly used in screening tests proved too slow for use with the pink root fungus, Pyrenochaeta terrestris. On this account preliminary tests were made with the filter paper disc method which had been developed for the assay of the potency of some antibiotics, notably penicillin. The results obtained were promising, and appropriate modifications were worked out to permit its adaptation to the problem at hand. As finally standardized the modified method was relatively simple, required only 36 hours for completion, yielded quantitative results and apparently involved little or no decomposition of the chemicals.

Procedure

A pathogenic isolate of P. terrestris was used to inoculate 500-ml Erlenmeyer flasks containing 200 ml of modified Czapek's medium (2). Four 1-cm squares cut from the perimeter of colonies grown on modified Czapek's medium solidified with 2% agar were used to inoculate each flask. The flasks were set up on a laboratory table for 12 days at room temperature and were shaken daily for the first 8 days to insure sufficient aeration. The contents of each flask were removed at the end of this time and macerated for 1 minute in a Waring Blendor. The macerated mycelium was used to seed the agar at the rate of 25 ml of mycelial suspension per 100 ml of agar. One half gram of streptomycin sulfate (740 mcg per mg) was added to each liter of agar to prevent the growth of bacterial contaminants.

Solutions or suspensions of the fungicides and chemotherapeutants to be tested were prepared and standard filter paper discs[3] were soaked in them for 30 minutes. The discs were then re-

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[1]Portion of a dissertation presented by the senior author to the Graduate School of Louisiana State University in partial fulfillment of the requirements for the Ph. D. degree in plant pathology.

[2]Respectively, First Lieutenant, U. S. Army Biological Warfare Laboratories, Fort Detrick, Maryland, formerly Graduate Research Assistant; and Professor of Plant Pathology, Louisiana State University, Baton Rouge, Louisiana.

[3]12.7 mm diameter, supplied by Carl Schleicher & Schuell Co., Keene, N. H.

moved from the solutions, drained, and placed in a 30° C incubator to dry.

After the inoculum and streptomycin sulfate were added to the cooled agar, the plates were poured and allowed to solidify. Then the filter paper discs were applied to the agar surface, two discs per plate, two plates for each material to be tested. The plates were put in a 27° C incubator for 36 hours and at the end of that time the zone of inhibition around each disc was measured.

The diameter of the zone of inhibition varied directly with the magnitude of the inhibiting effect of the fungicide or chemotherapeutant tested by the filter paper disc method. However it was noted that the lack of inhibition demonstrated by certain fungicides and chemotherapeutants might be due to their inability to diffuse through agar.

In a preliminary test preparations of certain wettable fungicides would not exhibit any inhibitory effects unless the surface of the disc bearing the deposit was placed face down on the agar in the plate. Accordingly, all discs in these tests were placed with the deposit side face down (Fig. 1).

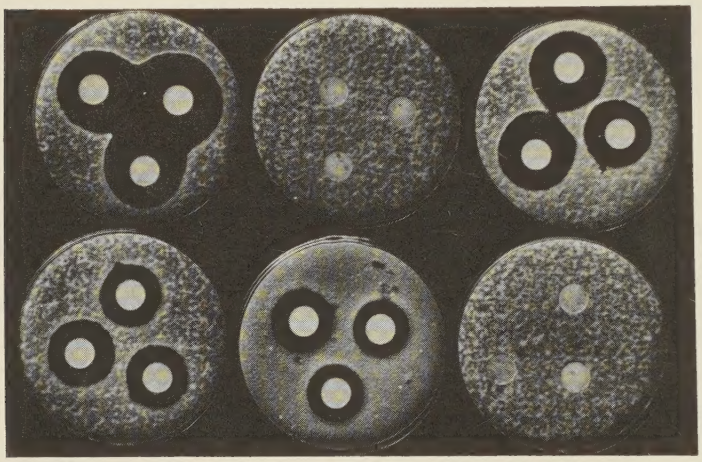

FIGURE 1. Zones of inhibition produced in a modified filter paper disc test[4] after 36 hours. Materials used at 1000 ppm. Top left: Mema MN; center: Acti-dione-acetate derivative; right: Panogen 15; Bottom left: B. B. 67; center: Mercuric chloride; right: control (water).

Results

Fifty-four fungicides, many of them newly released for experimental study, were tested at 1000 ppm using the modified filter paper disc technique. The results of these tests are presented in Table 1. A zone diameter of 12.7 mm, the diameter of the filter paper disc, signifies no inhibition. Of the 54 fungicides tested, 25 produced highly significant inhibition of P. terrestris while two others produced significant results.

The fungicides tested in the laboratory at 1000 ppm were later tested in sand culture in the greenhouse at the same concentration. Twenty of these fungicides which prevented the development of pink root in sand culture were then tested at 200 ppm in the laboratory. Only three of these materials, namely Omadine-Thiourea salt, Omadine-Zinc salt, and Omadine-Disulfide salt, caused significant inhibition of P. terrestris when tested by the modified filter paper disc technique at this lower concentration.

Twenty-nine chemotherapeutants were tested at 1000 ppm for their ability to inhibit P. terrestris using the modified filter paper disc method. Of these, CP 8525, CP 10616, CP 10512 (Monsanto), Carbide and Carbon Experimental Compound 1182, Mycostatin (Olin Mathieson), Agrimycin 500 (Pfizer), Anisomycin (Pfizer), and Acti-dione (Upjohn) caused highly significant inhibition of P. terrestris while CP 8621, CP 9425, Achromycin hydrochloride, Neomycin sulfate, Agrimycin 100, Olin Mathieson Chemical #G-1143, 8-OH-Quinoline-sulfate, 8-Quinolinol-sulfamate, potassium penicillin, streptomycin sulfate, Oligomycin, Pfizer 165, Terramycin hydrochloride, Aureomycin, Acti-dione-acetoacetate derivative, Acti-dione-thiosemicarbazone derivative, Acti-dione-methyl hydrazone derivative, Acti-dione-acetate derivative, Acti-dione-semicarbazone derivative, Acti-dione-oxime derivative, and griseofulvin caused nonsignificant inhibition of the pink root fungus.

[4]Three discs were used in the first series of preliminary tests.

Table 1. Average diameter of the zone of inhibition (in mm) using Pyrenochaeta terrestris in modified filter paper disc tests with a number of fungicides[a].

Material	Zone diameter[b] (in mm)
TEST 1	
Mema MN (15%) (Chipman Chemical Co.)	39.0
Mema RM (4.88%)	34.4
B. B. 67 (1.85%) (Chipman Chemical Co.)	39.9
Calo-Clor (Mallinckrodt)	26.3
Omadine, Manganese salt (Olin Mathieson)	49.6
Omadine, Disulfide salt	52.3
Omadine, Zinc salt	28.8
Omadine, Thiourea salt	51.4
Omadine, Ferric salt	19.8
Water Check	12.7[c]
L.S.D. 0.05 5.55	
L.S.D. 0.01 8.41	
TEST 2	
Omadine, Sodium salt	51.3
Omadine, Copper salt	24.3
WO 4778 (1%) (Panogen)	46.1
Panogen 15 (2.2%)	54.3
Pittsburgh B-1843 (Chemagro Corp.)	31.3
Chemagro D-113	40.3
Chemagro D-121	34.6
Kromad (Mallinckrodt)	28.3
Water Check	12.7
L.S.D. 0.05 9.03	
L.S.D. 0.01 13.68	
TEST 3	
Crag. Exp. Fung. 5400 (Carbide & Carbon)	28.6
Thioneb 50W (U. S. Rubber)	29.9
Crag Exp. Fung. 224	22.3
MM 21 (Mallinckrodt)	19.8
Water Check	12.7
L.S.D. 0.05 4.80	
L.S.D. 0.01 7.27	
TEST 4	
MSG 40 (Mallinckrodt)	22.6
MSG 1	32.3
Ortho Phaltan 50W (Calif. Spray-Chemical Corp.)	17.8
Water Check	12.7
L.S.D. 0.05 1.35	
L.S.D. 0.01 2.04	
TEST 5	
Dithane + $TiCl_2$[d]	16.5
Dithane + $ZnSO_4$	18.6
Dithane (Rohm & Haas)	16.1
Water Check	12.7
L.S.D. 0.05 2.15	
L.S.D. 0.01 3.26	

[a]The following fungicides produced nonsignificant results: Hexachlorobenzene, 8-OH-7-Iodoquinoline-5-Sulphonic Acid, American Cyanamid Exp. Fung. 5223, 12607, and 23441, PVP Iodine, Dow Compounds M-244 and M-245, Cadminate, Puratized 1143 and 1180, RE 4334, Merck Organic Cadmium Fung. H258A, Stauffer Compounds N-244 and N-521, MSG 2, MSG 3, MSG 4, Dithane + $SnCl_2$, Dithane + $CrKSO_4$, Dithane + $CeNO_3$, Dithane + $ZiNO_3$, Dithane + $NiCl_2$, Dithane + $CuSO_4$, Dithane + $HgCl_2$, maneb, Chemagro Compound C-272.

[b]Average of four replications. [c]Diameter of paper disc, indicates no inhibition.

[d]Formulated to contain an amount of metal equal to that in 1000 ppm of maneb (70% active).

GREENHOUSE SCREENING

Procedure

Pots of infested sand were prepared by mixing 200 ml of inoculum plus 500 ml of Hoagland's solution (2) with 10 kg of sand. The inoculum was obtained by growing isolates of P. terrestris in modified Czapek's medium for 12 days, and then macerating the cultures for 1 minute in a Waring Blendor. Each of 4-inch pots was treated with a 100-ml drench of a solution of one of the fungicides or chemotherapeutants and onion seed were sown immediately and covered with clean sand. Each pot was planted with 50 seeds of the pink root susceptible onion variety White Sweet Spanish. The pots were watered daily and Hoagland's solution was added once a week. At the end of each test, the plants were carefully removed from the pots, weighed, counted, and graded for disease severity.

Results

A number of fungicides and chemotherapeutants previously studied in the laboratory were tested in sand culture at 1000 ppm. None of the 29 chemotherapeutants reduced the incidence of pink root to 1% or less; however 22 of the 50 fungicides tested did reduce the disease index to 1% or less. These 22 fungicides were Mema MN (1.5%; 200 ppm.), RE 4334 (500 ppm; California Spray-Chemical Corporation), B. B. 67 (1.85%), Omadine-Copper salt (phytotoxic), Mema RM (4.88%), Panogen 15 (2.2%), Thioneb 50W, Omadine-Manganese salt, Omadine-Zinc salt, Omadine-Disulfide salt, Crag Experimental Fungicide 5400 (phytotoxic), Chemagro C-272, Omadine-Thiourea salt (200 ppm), Pittsburgh B-1843, Chemagro D-113 (100 ppm), Dow Compound M-244, Dow Compound M-245, Crag Experimental Fungicide 224 (phytotoxic), Dithane plus titanium chloride[5], Dithane plus cerium nitrate, Dithane plus zirconium nitrate, and Dithane. The four materials previously tested at 1000 ppm in the laboratory but not tested at that concentration in sand culture were Stauffer Compound N-521, MSG 40, MSG 1, and Dithane plus nickel chloride.

Twenty of the 22 fungicides[6] which had performed well in sand culture at 1000 ppm were retested by this method at 200 ppm. Nine of these 20 materials reduced the incidence of pink root to 1% or less: Mema MN (1.5%, 40 ppm), B. B. 67 (1.85%), Omadine-Manganese salt, Omadine-Disulfide salt, Omadine-Thiourea salt, Omadine-Zinc salt, Thioneb 50W, Pittsburgh B-1843, and Dithane.

FIELD TESTS

A number of experiments were set up in 1957 and 1958, in which the most promising fungicides were used in efforts to control pink root of shallots. The disease did not develop in sufficient amounts in any of the tests to give conclusive results.

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DEPARTMENT OF PLANT PATHOLOGY, LOUISIANA STATE UNIVERSITY, BATON ROUGE, LOUISIANA

[5]The Dithane materials were formulated to contain an amount of metal equal to that in 1000 ppm of maneb (70% active).

[6]Crag Experimental Fungicide 5400 and Dithane plus zirconium nitrate were omitted from these tests.

PROMISING DECAY INHIBITORS FOR POSTHARVEST USE ON FLORIDA ORANGES

J. J. Smoot, G. A. Meckstroth, and C. F. Melvin[1]

Summary

One hundred and twenty-four compounds obtained from the citrus fungicide-screening program were further tested. Twenty of these compounds reduced postharvest decay as much as 75% after 2 weeks' storage at 70° F. In direct comparison, three compounds (carbanilic acid, m-chloro-, isopropyl ester; hydracrylonitrile, carbanilate; and 2-propyn-1-ol, carbanilate) were more effective in all tests than sodium orthophenylphenate + hexamine. Water-wax emulsions of two of these compounds gave promising results.

Since 1946 about 3000 fungicides have been tested for inhibition of postharvest decay of citrus fruit at the United States Department of Agriculture's Horticultural Field Station at Orlando, Florida. Results of these screening tests and the methods used have been reported previously (3, 4, 5, 6). Relatively few of the compounds tested were effective against the major postharvest decays of Florida citrus which include stem-end rot (Diplodia natalensis Pole-Evans and Phomopsis citri Fawc.) and green mold (Penicillium digitatum Sacc.).

This report summarizes the results of larger scale tests of the more promising compounds. These compounds may have potential commercial value for use on citrus or other fruits and vegetables.

MATERIALS AND METHODS

The present studies were conducted on naturally infected, legally mature Florida oranges. Varieties such as Parson Brown and Hamlin, Pineapple, and Valencia produced on mature trees were used. Most fruit was harvested from commercial groves in Central Florida. Where practical, fruit for successive tests was harvested from the same trees. All fruit was harvested by clipping to insure that the "button" or calyx was left attached. The incidence of stem-end rot during storage has been shown to be higher when the buttons are present (2).

All lots were washed in a commercial-type fruit washer. The fruit was then randomized into either 50- or 100-fruit samples for treatment. During the first half of the season (October to February), the fruit that would normally be degreened for commercial use was placed in the "coloring" room and treated with ethylene at approximately 85° F and 90% relative humidity for periods of 12 to 72 hours, depending on the natural color of the fruit. In several tests conducted during the latter part of the season (March to May) test fruit was also pretreated with ethylene to determine the effect of the compounds on ethylened as well as non-ethylened fruit.

Within a few hours after washing and drying, or after leaving the "coloring" room, the oranges were treated by dipping them in fungicidal solutions at room temperature for a specified length of time. They were then dried either by exposure to open air or in later tests on a commercial-type forced warm-air fruit drier. Samples that were spray rinsed with tap water following dipping remained in the fungicide about 2 minutes. Fruit to be left unrinsed usually was given a 10-second dip. After drying, the oranges were placed in open field boxes and stored for 3 weeks at 70° F. Oranges were inspected at weekly intervals and the numbers of fruit showing decay, chemical injury, and physiological rind breakdown were recorded. One hundred and twenty-four compounds and preparations that had reduced decay in the screening program were tested. Various formulations and methods of application were used. Water-soluble chemicals were used in aqueous solution; otherwise, various organic solvents such as ethanol and acetone were used. In certain tests, water emulsions and dispersions were prepared. In others, the chemicals were added to the water phase of wax emulsions.

During the season of 1958-59, the 10 compounds which were still available from the manufacturers were retested (Table 1). These tests were conducted in the same manner as in previous tests, except that direct comparisons were made with sodium orthophenylphenate + hexamine (Dowicide-A + hexamine, the standard postharvest decay inhibitor recommended for citrus). Fresh solutions of the test chemicals and of Dowicide-A + hexamine were prepared for each test. All lots were spray rinsed following treatment.

[1]Plant Pathologist, formerly Plant Pathologist, and Biological Aid, respectively, Market Quality Research Division, Agricultural Marketing Service, United States Department of Agriculture, Orlando, Florida.

Table 1. Decay-inhibiting properties of 10 promising compounds in comparison with sodium orthophenylphenate + hexamine on oranges stored at 70° F, 1958-59 season.

Name of compound	Concentration (%)	Solvent	Season	Ethylened	% decay reduction[a] after:		% decay reduction in comparable oranges treated with Dowicide-A + hexamine[b] after:	
					2 weeks	3 weeks	2 weeks	3 weeks
Carbanilic acid, m-chloro-, isopropyl ester	5	95% Ethanol	Fall	Yes	44	22	0	0
do.	5	95% Ethanol	Winter	No	68	61	71	40
do.	5	95% Ethanol	Spring	No	89	89	79	51
Carbanilic acid, 1-methoxy-, 2-propyl ester	2 1/2	95% Ethanol	Fall	Yes	45	13	0	0
do.	2 1/2	95% Ethanol	Winter	No	54	30	71	40
Carbanilic acid, 2-methoxy-, propyl ester	2 1/2	95% Ethanol	Winter	Yes	52	21	40	22
do.	2 1/2	95% Ethanol	Winter	No	37	10	50	32
Hydracrylonitrile, carbanilate	5	47% Ethanol	Winter	No	82	84	59	26
do.	5	47% Ethanol	Fall	Yes	95	84	30	10
do.	5	47% Ethanol	Winter	No	86	81	86	59
do.	5	47% Ethanol	Spring	No	95	91	79	51
2-Propyn-1-ol, carbanilate	5	95% Ethanol	Winter	Yes	92	69	54	15
do.	5	95% Ethanol	Winter	No	84	79	76	45
do.	5	95% Ethanol	Spring	No	95	91	79	51
8-Quinolinol condensation product	4	32% Ethanol	Winter	Yes	44	22	40	22
do.	4	32% Ethanol	Winter	No	10	0	50	32
8-Quinolinol sulfate	5	Water	Fall	Yes	15	12	30	10
do.	5	Water	Winter	No	71	53	86	59
Thioacetamide	5	Water	Fall	Yes	7	8	30	10
do.	5	Water	Winter	No	67	-13[c]	86	59
Urea-boric acid reaction product #1	5	Water	Fall	Yes	10	- 6[c]	48	12
do	5	Water	Winter	No	31	29	79	59
Urea-boric acid reaction product #2	5	Water	Fall	Yes	5	- 9[c]	48	12
do.	5	Water	Winter	No	38	29	79	59

[a]As compared with untreated control; each value based on two tests of approximately 100 fruit each.
[b]Concentration of Dowicide-A + hexamine = 2% + 1%.
[c]Minus sign indicates an increase in decay as compared with untreated control.

RESULTS AND DISCUSSION

Of the 124 compounds from the screening program that were intensely tested, 20 were considered to be the most promising. Each of these 20 selected compounds reduced total decay at least 75% after 2 weeks' storage at 70° F in one or more tests, and caused no more than slight to moderate chemical injury. The compounds are as follows:

Carbamic acid, thiono-, isopropyl ester
Carbanilic acid, p-chloro-, ethyl ester
Carbanilic acid, m-chloro-, isopropyl ester
Carbanilic acid, 2-methoxy-, propyl ester
Carbanilic acid, 1-methoxy-, 2-propyl ester
Carbanilic acid, propyl ester
Carbanilic acid, thiono, p-methoxy-, ethyl ester
Hydracrylonitrile, carbanilate
8-Hydroxyquinoline benzoate
8-Hydroxyquinoline sulfate
Methoxybutenyl carbanilate mixture
2-(2-Methoxybutenyl)ethyl carbanilate
Propyl benzoyl-thionocarbamate
2-Propyn-1-ol, Carbanilate
8-Quinolinol condensation product
8-Quinolinol sulfate
Thioacetamide
m-Toluidine, 4-ethylsufonyl-, a, a alpha-trifluoro
Urea-boric acid reaction product (1 boric acid to 1 urea)
Urea-boric acid reaction product (2 boric acid to 1 urea).

In most instances the compounds were more effective on non-ethylened than on ethylened fruit. This can be explained by two factors: the warm-temperature delay of 1 and 3 days from harvest to time of treatment; stimulation of growth of D. natalensis by ethylene (1).

Chemical injury was manifested as a discoloration and collapse of the rind tissue. Physiological rind breakdown (aging and pitting) was not consistently associated with any of the chemical treatments.

Several chemicals including thiourea and various mercurials gave excellent control but have not been included above because of their known toxicity to warm-blooded animals. Compounds which the manufacturers have reported as too expensive to synthesize have not been included.

The results of tests conducted during the 1958-59 season (Table 1) show that three compounds (carbanilic acid, m-chloro-, isopropyl ester; hydracrylonitrile, carbanilate; and 2-propyn-1-ol, carbanilate) consistently gave better results than Dowicide-A. Three other compounds (carbanilic acid, 1-methoxy-, 2-propyl ester; carbanilic acid, 2-methoxy-, propyl ester; and 8-quinolinol condensation product) gave better results than Dowicide-A in the fall tests but were not as effective on midseason fruit tested during the winter. The other four compounds were not as effective as Dowicide-A. None of the 10 compounds retested during 1958-59 caused chemical injury of commercial importance, and none appeared to increase the amount of physiological rind breakdown.

Eleven of the 20 chemicals already enumerated are derived from carbanilic acid. No explanation is offered as to why this group of compounds is so effective against postharvest decay of citrus, particularly stem-end rot. Several of these derivatives, however, have been shown to be effective as herbicides for crop use.

Table 2. Effect of two carbanilates and Dowicide-A + hexamine in water-wax emulsions on decay of Valencia oranges.

Treatment	Concentration (%)	Number of fruit	% decay reduction after: 2 weeks at 70° F	% decay reduction after: 3 weeks at 70° F
Hydracrylonitrile, carbanilate	1	109	92	77
2-Propyn-1-ol, carbanilate	1	111	100	91
Dowicide-A + hexamine	1 + .5	111	74	21

The three most effective compounds are not sufficiently water soluble to be of commercial value as a dip or flood treatment when dissolved in water. The cost of ethanol and other organic solvents precludes their use on a commercial scale. Preliminary testing in the spring of 1959 has indicated that two of these materials can be successfully formulated into a water-wax emulsion and applied in that form (Table 2). These two carbanilates reduced decay by 92 and 77% and 100 and 91% after 2 and 3 weeks, respectively. In comparison, Dowicide-A + hexamine reduced decay by 74 and 21%. There was no chemical injury or increased rind breakdown from treatment with either of these chemicals.

The possible use of wax emulsions as carriers for these compounds will be explored further during the 1959-60 harvest season.

The chemical compounds discussed in this paper are not recommended for use as post-harvest fungicides on citrus or other fruits or on vegetables at this time. The toxicity of these materials to warm-blooded animals has not been determined; nor have residue tolerances been established by the Food and Drug Administration, Department of Health, Education, and Welfare.

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MARKET QUALITY RESEARCH DIVISION, AGRICULTURAL MARKETING SERVICE, UNITED STATES DEPARTMENT OF AGRICULTURE

RELATIONSHIP OF HOJA BLANCA TO THE INOCULATION POINT AND TO THE AGE AND YIELD OF RICE PLANTS

William W. McMillian[1], Judson U. McGuire[1], and H. A. Lamey[2]

Summary

The hoja blanca disease has become a serious threat to rice in the United States. Sogata orizicola Muir, the sole known insect vector, transmits the virus with varying results depending on the age of the rice plant and the point of inoculation. When viruliferous insects were fed at three locations on plants of a susceptible variety of rice ranging from 2 weeks to 3 months old, the symptom appeared in 6 to 34 days. In field tests with susceptible varieties, loss in panicle weight ranged from 22 to 82%, depending upon the age of the plant when infection occurred. Total loss in rice from diseased plants is increased by low milling quality.

INTRODUCTION

Hoja blanca or white leaf, an insect-transmitted virus disease of rice, has received widespread attention during the last 3 years. This disease, destructive in many Latin American countries, has been found in the United States (3, 4) and is considered a threat to rice in this country (5).

It is difficult to estimate the amount of damage that can be attributed to hoja blanca. Sogata orizicola Muir is the sole known vector (2). The vector transmits the pathogen with varying results, depending on the age of the rice plant and/or the part of the plant where it feeds. Young plants may be severely stunted or killed but plants infected when near maturity suffer only minor detrimental effects.

Rice plants infected with the hoja blanca virus can usually be detected easily in the field. If young rice plants are fed upon by viruliferous insects, symptoms of the disease appear in the plants in a short time. The first sign is a chlorotic mottling in the emerging leaf or in the area of elongation of the next youngest leaf. As the plant develops the new leaves become progressively more chlorotic.

Plants that reach maturity will be stunted (1), if they are infected in an early stage of growth. The panicles will be reduced in size, and the lemma and palea will be distorted and discolored. The flag leaf and two or three of the next older leaves will have chlorotic striping and the lower leaves will usually be completely chlorotic. A plant that is infected when about 2 months old will have chlorotic mottling and striping of the leaves that develop thereafter. The plant will be stunted and the panicles distorted, but not so much as in early-infected plants. Late infection usually causes only the flag leaf and sheath to be chlorotic, and the degree of panicle distortion depends upon the time and place of initial infection.

Although there is considerable overlapping of symptoms, in this paper symptoms will be grouped according to the age of the plants: 1) young plants; 2) plants of intermediate age; and 3) nearly mature plants.

The purpose of this paper is to discuss the time variance for hoja blanca symptoms to appear in a plant, depending on the age of the plant and the point of inoculation, and the eventual effects that these variables have on the rice yields. All experiments were conducted at the Hoja Blanca Research Laboratory near Camaguey, Cuba.

PROCEDURE

Insects were fed at three locations on plants of three different ages. The feeding locations were emerging leaf, second youngest leaf, and the leaf sheath at the plant base, and the plant ages were 2 weeks, 1 month, and 3 months. The susceptible variety Nato was used. Each treatment consisted of a pot containing two rice plants of the same age and 10 insects feeding at one location. There were 20 replications of each treatment.

To determine the loss in yield depending on the time of inoculation, plants growing in 0.1-

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acre plots of the varieties Nato, Bluebonnet 50, Century Patna 231, and Toro were observed in the field at the Camaguey Station. The rice plants were inoculated with a natural population of Sogata orizicola. Virus symptoms were noted as they appeared on each plant. The degree of loss in yield, depending on the time of inoculation, was determined by collecting 100 panicles from plants in each of the three aforementioned age groups for each variety. The average weight of panicles for each time-of-symptom-appearance was determined.

To determine the milling loss in diseased panicles, a 100-gram sample of each variety, composed of plants in the three age groups mentioned before, was run through a McGill cleaner and sheller, and the loss in weight was compared with similar samples from healthy rice.

In order to project a loss in yield on a per-acre basis under natural conditions existing at the Camaguey station in 1959, a 0.5-acre plot of Nato was sown in an area known to be infested with S. orizicola. Usual cultural practices were employed. At the time of harvest all plants were counted and examined for symptoms of hoja blanca. Nine hundred panicles of diseased rice composed equally from the three plant age groups were collected and weighed. The average weight of these panicles was compared with the average weight of a sample of healthy panicles from the same plot.

RESULTS

As shown in Table 1, there was a marked variance in the time for symptom appearance. The older the plant the longer it took for symptoms to appear. Within any age group, symptoms appeared first when feeding was on the emerging leaf. As insects fed on progressively older tissues within each plant age group, the length of time for symptoms to appear increased, except in the 3-month-old group, which showed no difference between the second and third feeding positions. The time range was from 6 to 34 days.

Table 1. Average number of days required after inoculation for hoja blanca symptoms to appear on different aged Nato rice plants fed on at three locations.

Location	2 weeks	1 month	3 months
Emerging leaf	7	18	26
Second youngest leaf	10	21	34
Leaf sheath at plant base	14	26	34

Data reported in Table 2 show that greater reduction in yield occurred in plants infected at an early age. Those early infected plants that reached maturity produced little or no grain. Stunting and shading probably contributed to the low yield. Medium-age plants that were infected were stunted. Some sterility apparently occurred as many flower parts on diseased plants were malformed and the grain was of poor quality from flowers that formed. Late infected plants exhibited small losses in the amount produced, and the quality was often poor.

Table 2. Effect of variety and age of plant, when hoja blanca inoculation occurs, on weight of rice panicles[a].

Age	Panicle weight (in grams)			
	Nato	Toro	Century Patna 231	Bluebonnet 50
Young	1.2	0.9	1.2	1.0
Medium	2.8	1.0	2.1	2.9
Nearly mature	3.9	2.4	1.4	3.9
Check (not infected)	5.0	5.1	4.2	5.0

[a]Average of 100 panicles.

Table 3 shows the loss in weight of four diseased varieties after cleaning and milling. This loss can be attributed mostly to distorted and poorly formed kernels. Bluebonnet 50 exhibited the most severe loss during milling, and Century Patna 231 the least.

In the 1/5-acre plot of Nato, 35% of the panicles were diseased. The average weight of diseased panicles was 2.7 grams compared with 5.0 grams for healthy panicles. Thus, the average weight of all panicles in this plot was 4.2 grams or a reduction in yield of 16% caused by the disease. Based on the results shown in Table 3, it is estimated that cleaning loss and

Table 3. Effect of hoja blanca on the percentage loss in weight of the grain of four rice varieties after being cleaned and/or milled[a].

Variety	After cleaning		After milling	
	Healthy	Diseased	Healthy	Diseased
Century Patna 231	2	9	23	30
Nato	2	19	25	42
Toro	7	16	25	48
Bluebonnet 50	6	52	27	89

[a]Appreciation is extended to Mr. Raul Silva, in charge of the Molino Arrocero Jayamá, S. A. Laboratory for his assistance in cleaning and milling.

lower milling quality of the grain from diseased panicles would cause an additional loss of about 13%.

CONCLUSIONS

The data presented show that early infected plants are more severely injured than late infected plants. The time required for symptom development was directly proportional to the age of the plant when inoculated. When plants were inoculated at three points and at three ages, the length of time for development of symptoms varied to a great extent. Some of the varieties of rice studied were more severely damaged by the hoja blanca virus than others, although there was an appreciable loss in yield in all diseased plants.

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ENTOMOLOGY RESEARCH DIVISION AND CROPS RESEARCH DIVISION, AGRICULTURAL RESEARCH SERVICE, UNITED STATES DEPARTMENT OF AGRICULTURE

OCCURRENCE OF HOJA BLANCA AND ITS INSECT VECTOR, SOGATA ORIZICOLA MUIR, ON RICE IN LOUISIANA

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Summary

Surveys were made for hoja blanca and its insect vector, Sogata orizicola Muir, in Louisiana rice fields in 1959. Both were found in a number of fields in several parishes for the first time in Louisiana and for the first time in major rice-producing areas of the United States.

Hoja blanca (white leaf), a potentially destructive rice disease, was found in a number of Louisiana rice fields in 1959. The disease had been found in Florida in 1957 (2) and in south Mississippi in 1958 (3).

Sogata orizicola Muir, a fulgorid, is the vector of the virus causing hoja blanca (1, 4). This insect was not reported for the United States until 1957, when the disease was found near Belle Glade, Florida.

As the finding of hoja blanca near Belle Glade, Florida in a limited rice-producing area represented the first occurrence of a potentially serious disease in the United States, eradication measures were initiated by the Plant Pest Control Division, United States Department of Agriculture, and the Florida State Plant Board. Similar control measures were employed in 1958 upon finding the disease and insect vector in Hancock County, Mississippi. The 1958 disease and vector surveys were negative in Arkansas, Louisiana, Texas, and Mississippi except for the two locations in Hancock County.

RESULTS OF 1959 SURVEYS

Extensive cooperative surveys were planned for 1959 in a meeting held at Beaumont, Texas on June 16, 1959, attended by representatives of the United States Department of Agriculture, State experiment stations, and state regulatory agencies of the four southern rice-producing States. Later, the surveys were initiated by personnel of the Plant Pest Control Division in cooperation with the other agencies in each of the four States. The field survey crews collected insects from rice with sweep nets and preserved questionable specimens, chiefly fulgorids, for examination by an insect taxonomist. Fields with rice plants exhibiting disease symptoms similar to those of hoja blanca were checked by a plant pathologist who examined leaf specimens or made later inspections of the field. While abnormal plants with leaf coloration similar to that associated with hoja blanca were frequently found, definite identifications were based on only typical, advanced foliage symptoms.

On August 3 hoja blanca was identified in three rice fields in St. Tammany Parish near Covington, Louisiana, in which S. orizicola was collected on July 23. St. Tammany is adjacent to Hancock County, Mississippi, where the disease and vector had been found in 1958.

After the finding of hoja blanca and S. orizicola in St. Tammany Parish, the rice-producing section along the Mississippi River was extensively surveyed. As shown in Table 1, the vector was first collected in this area on August 10 and then later in each of the parishes. Later in the season S. orizicola was found in St. Landry, Evangeline, St. Martin, and Vermilion parishes in southwest Louisiana and in Madison Parish in the northeast part of the State.

As shown in Table 1, S. orizicola was collected in 14 parishes and hoja blanca was identified in 11 of these. The disease and vector were always associated providing plant-growth conditions were favorable for symptom development. Failure to find hoja blanca in three of the parishes, Evangeline, Vermilion, and St. Martin, was attributed to unfavorable conditions late

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Table 1. Louisiana parishes where hoja blanca and Sogata orizicola were found in 1959.

Part of State and Parish	1959 Rice acreage[a]	Date of initial finding: S. orizicola	Date of initial finding: hoja blanca	Fields infested: number	Fields infested: acreage
Southeast					
St. Tammany	150	July 23	Aug. 3	3	135
River and Teche					
St. John	720	Aug. 10	Sept. 10	2	651
St. James	2,968	Aug. 12	Sept. 10	9	2,672
Iberville	1,677	Aug. 14	Sept. 11	5	1,224
Lafourche	395	Sept. 11	Sept. 24	1	206
Terrebonne	146	Sept. 11	Sept. 24	1	69
Assumption	531	Sept. 15	Sept. 24	2	131
Ascension	1,696	Oct. 1	Oct. 27	1	245
St. Mary	2,866	Sept. 28	Oct. 27	4	558
St. Martin	3,547	Nov. 2	[b]	3	363
Southwest					
St. Landry	15,276	Sept. 23	Sept. 25	11	1,418
Evangeline	39,940	Oct. 2	[b]	5	253
Vermilion	102,964	Oct. 22	[b]	1	60
Northeast					
Madison	775	Oct. 16	Oct. 28	1	60
Total	173,651			49	8,045

[a]Rice acreage in the United States, 1959. Rice Journal 62(10): 28-29. 1959.
[b]Hoja blanca not found.

in the season. In late October plant growth was scanty because of grazing by cattle as well as unfavorable growing conditions. Many of these were stubble fields.

Figure 1 shows 14 infested parishes listed in Table 1 in relation to the principal rice-growing areas of Louisiana, Arkansas, Mississippi, and Texas. Two infested counties in Mississippi, Hancock and Harrison, and Palm Beach County, Florida are also included.

The objective of the survey was to determine at the earliest possible time the distribution of hoja blanca and the vector in the southern rice area. Experience early in the survey indicated that the vector can be found more readily than the disease. Theoretically, and on the basis of observations on the disease and insect counts in several fields, only a few insects, or possibly only a single viruliferous female, migrate or are blown into a field. Later, one or more small areas 10 to 15 feet across with a fairly high percentage of diseased plants and relatively high insect populations, particularly nymphs, are found in the field. Such limited infected areas are difficult to locate in 40- to 200-acre fields and constitute a practical limitation to routine surveys of a large number of fields. But, by the time hoja blanca symptoms are well defined, even though confined to limited areas within the field, the insect vector has completed one or possibly two life cycles and spread over much of the field. Thus, chances of finding the vector were better than finding the disease in routine surveys of large fields. For this reason and because of some difficulty encountered by field survey crews in identifying disease symptoms, emphasis was placed on vector surveys. The fields with S. orizicola were later examined for the disease. As a result the vector was collected prior to finding the disease, as shown in Table 1.

On the basis of planthopper populations and stage of development of disease symptoms, the field of initial infestation in Louisiana was probably in St. Tammany Parish. As the hoja blanca symptoms observed on August 3 appeared on the two top (youngest) leaves and panicles, infection probably occurred in early June. The distribution of diseased plants and planthopper population indicated that the vector had completed two generations, each of approximately 30 days, by August 3. Several of the fields in other parishes showed a pattern of scattered diseased plants which indicated the initial entry of several viruliferous planthoppers.

After collection of S. orizicola in rice fields in St. Tammany Parish and each of the other fields, insecticides were applied by airplane at fairly heavy rates. Phosdrin (1-methoxycarbonyl-1-propen-2-yl-dimethylphosphate) at 0.5 or a mixture of malathion at 1 pound plus DDT at 1 pound per acre was used, depending upon stage of maturity of the rice. The spray applications were continued at 10-day intervals as long as any Sogata specimens were collected a few days after spraying. In most of the fields none could be collected 1 or 2 days after spray-

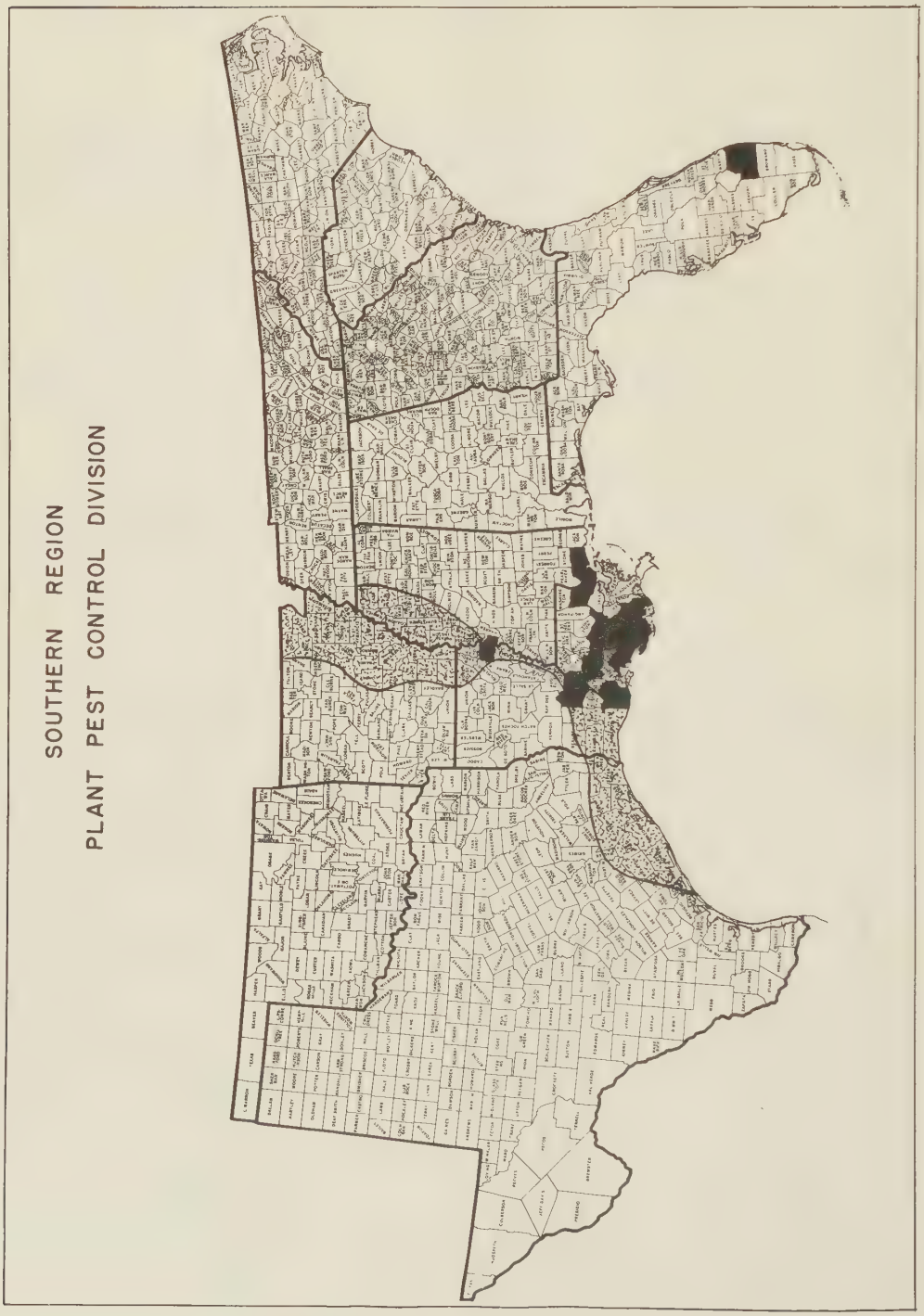

FIGURE 1. Relation of infested areas (in black) to the principal rice-producing area in southern States.

ing, but a few appeared several days later necessitating additional applications. The insecticides gave excellent practical control but were not satisfactory for eradication.

DISCUSSION

Environmental conditions during the summer and fall in Louisiana are favorable for the multiplication and spread of S. orizicola. Observations indicate that the planthoppers became established in early June in rice in St. Tammany Parish and possibly also in the river section. The source of the infestation is not known. Although the best known methods for controlling the vector were used, a few of the vectors were still present in some fields when killing frosts occurred, during the week of November 1. Whether this species is capable of surviving the winters in Louisiana is not known.

As with other pests capable of fairly rapid multiplication and spread and eluding early detection in newly infected fields, the control measures used for S. orizicola were not as successful as desired. Drastic reduction in population was achieved and it is believed the measures used prevented a more rapid spread of the insects.

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PLANT PEST CONTROL AND CROPS RESEARCH DIVISIONS, AGRICULTURAL RESEARCH SERVICE, UNITED STATES DEPARTMENT OF AGRICULTURE, LOUISIANA AGRICULTURAL EXPERIMENT STATION, AND LOUISIANA DEPARTMENT OF AGRICULTURE AND IMMIGRATION

TRANSMISSION OF HOJA BLANCA OF RICE BY THE PLANTHOPPER, SOGATA CUBANA[1]

Guillermo E. Galvez[2], H. David Thurston[3], and Peter R. Jennings[4]

Hoja blanca, a virus disease of rice, has been shown to be transmitted by the planthopper Sogata orizicola Muir[5,6]. In addition, large numbers of Sogata cubana (Crawf.) have been found in collections made from rice and certain weeds, especially Echinochloa colonum, which had symptoms similar to those of hoja blanca in rice. Transmission studies were initiated to determine if S. cubana is involved in the epidemiology of the disease. Methods similar to those reported in the study of transmission by S. orizicola were used[6].

Insects allowed to feed on diseased rice plants for 3 days were transferred to E. colonum in groups of 20, 10, 5, and 1. The results are given in Table 1.

Table 1. Transmission of hoja blanca from rice to E. colonum by S. cubana.

Insects per cage	Total number of: Insects	Cages	Cages with plants showing symptoms
20	100	5	2
10	50	5	1
5	25	5	0
1	82	82	12[a]

[a]These 12 successfully transmitting insects included 1 nymph, 4 males, and 7 females.

Twenty single insects have been used to date to test transmission from E. colonum to E. colonum. Four transmissions have resulted with female planthoppers.

Three hundred insects in groups of 10 and 20, having previously fed on infected rice, have been tested for transmission to rice with negative results.

Seventy-three individual insects have been tested for transmission from E. colonum to rice. No successful transmissions resulted.

These preliminary data indicate that S. cubana can transmit hoja blanca from rice to E. colonum and from E. colonum to E. colonum, but not from rice to rice or from E. colonum to rice. Also, E. colonum has been shown to be a host of the disease.

COLOMBIAN MINISTRY OF AGRICULTURE, AND THE COLOMBIAN AGRICULTURAL PROGRAM OF THE ROCKEFELLER FOUNDATION

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A BLIGHT OF GROUND CHERRY AND RUSSIAN ALMOND SEEDLINGS CAUSED BY GLOEOSPORIUM FRUCTIGENUM BERK.

R. M. Gilmer

Summary

A severe tip blight of young ground cherry and Russian almond seedlings was identified as caused by Gloeosporium fructigenum. The fungus was seed-borne and was sufficiently deep-seated in some seeds to withstand mild surface sterilization.

During the course of routine seed germination tests in the greenhouse, a severe tip blight and dieback developed in young seedlings of ground cherry (Prunus fruticosa) and Russian almond (P. tenella). About 10% of the ground cherry seedlings and nearly 75% of the Russian almond seedlings were ultimately killed.

Examination of affected seedlings revealed that the succulent stems were completely girdled by a firm, dry, reddish-brown necrotic lesion, which was usually located at or near the point of attachment of the cotyledons to the stem. The roots of the affected plants were normal in appearance and remained alive for several days after the growing points had completely wilted.

Platings from 10 affected plants resulted in isolation of the following organisms: Gloeosporium--8; Penicillium--3; Geotrichium--1; unidentified bacteria--3. Only the Gloeosporium isolates proved pathogenic to young sour cherry seedlings (P. cerasus); wound inoculations with Gloeosporium spore suspensions resulted in a firm, reddish-brown decay that girdled the stems and killed the growing points within 3 to 5 days.

Circumstantial evidence indicated that the inoculum of Gloeosporium had been seed-borne on seed lots of Russian almond and ground cherry. Through the courtesy of Dr. C. R. Ure, Morden Experiment Station, Morden, Manitoba, who had supplied the original seed lots, additional seeds of both Prunus species were obtained. In a small proportion of the seeds of both species, the pit (bony endocarp) enclosing the true seed had split along the suture.

Fifty seeds of ground cherry were removed from pits with split sutures and were surface sterilized for 2 minutes in a solution of clorox (sodium hypochlorite 5.25%) -95% ethyl alcohol-water (1: 5: 19) before plating on acidified potato-dextrose agar. Gloeosporium was recovered from two of the seeds.

The ten Gloeosporium isolates (eight from seedlings and two directly from ungerminated seeds) appeared identical in culture. On potato-dextrose agar (Difco) the zonate colonies were relatively slow-growing even at 70° to 75° F. The mycelium was initially white to buff, but gradually became a dark olive-grey in 96 to 120 hours. Abundant bright salmon-orange spores were produced in numerous acervuli that developed beneath the aerial hyphae. In Czapek's solution in shake culture, mycelial growth was scanty, but sporulation was extremely heavy, so that the solution became distinctly orange-tinged within 48 hours. Spore measurements (based on 50 spores) were: Czapek's solution, 15-23μ x 4-5μ, with a mean of 18.4μ x 4.6μ; on PDA, 11-20μ x 2-4μ, with a mean of 14.2μ x 3.1μ.

Spore suspensions, inoculated into wounds, resulted in girdling and death of young seedlings of chokecherry (P. virginiana), peach (P. persica) and apple (Malus sylvestris) in addition to hosts previously listed. On uninjured ripe tomato fruits numerous anthracnose-like lesions were produced, while uninjured apple fruits developed a slow firm, brownish-black decay. From these data it appears probable that the fungus should be referred to Gloeosporium fructigenum Berk., the imperfect stage of Glomerella cingulata (Ston.) Spauld. & Schrenk.

It seems quite probable that either spores or mycelium of the fungus achieved contact with the ground cherry and Russian almond seeds through the split suture of the pit as the pits were removed from the fruit pulp. Although there was no obvious macroscopic evidence of seed infection, the fungus had evidently penetrated the seed coat and become sufficiently well established in the cotyledonary tissue to withstand mild surface sterilization.

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CONTROL OF HAWTHORN LEAF BLIGHT[1]

Forrest C. Strong[2]

Hawthorn leaf blight, caused by Entomosporium thuemenii (Cke.) Sacc., produces small brownish to nearly black lesions along the veins of the leaves of many species of hawthorns. The pink flowered variety of English hawthorn, Paul's Scarlet, Crataegus oxycantha variety pauli is especially susceptible and is quite regularly defoliated by mid-August to mid-September.

Experimental testing of proprietary organic mercury spray compounds from 1946 to 1950 established the fact that three applications at the recommended concentration of 1 pint of Puratized Agricultural Spray to 100 gallons of water or 4 ounces of Phix to 100 gallons of water were effective in checking the disease satisfactorily with good leaf cover on the trees until October 1 (2). These organic mercury compounds were applied at 10-day intervals beginning about July 10.

From 1952 to 1956 the effectiveness of the antibiotic Cycloheximide (Acti-dione) was tested using the prepared wafer form which is marketed under the proprietary name Acti-spray[3]. Three applications of a 20 ppm concentration were used at 10-day intervals, beginning early in July. No foliar injury from this concentration was observed, although when used on young leaves in the spring as low as 5 ppm causes severe leaf injury. Very good control of the disease was obtained even when the first application was made as late as July 18.

Beginning in 1957, reductions in the concentrations of Acti-spray were tested. During three seasons (1957, 1958 and 1959) it was found that this antibiotic chemical used at 5 ppm is as effective as 20 ppm in protecting the foliage from infection (Fig. 1). In some trees where infection of leaves on the lower branches and on the abundant sprouts which are common to Paul's Scarlet hawthorn had already caused considerable leaf fall, very few more leaves were lost (Fig. 2). Three spray applications were used, at 14-day intervals beginning about July 15.

Table 1. Percentage retention of foliage of Paul's Scarlet hawthorn sprayed for control of leaf blight.

	1957	1958	1959
Number of trees per treatment	8	11	16
Date of first application	July 17	July 14	July 11
Average foliage retention first spray date	100	95	100
Average foliage retention on October 1			
20 ppm Acti-spray	95	90	--
10 ppm Acti-spray	92	--	--
5 ppm Acti-spray	95	90	92
Control (unsprayed)	5[a]	2[a]	8[a]

[a]Leaves present only in tops of trees, lower branches and basal sprouts bare of leaves.

The trees were of different ages varying from 5 years in a plantation to 20 years in campus and boulevard ornamental plantings. Each sprayed and control group contained trees of both ages. Sprays were applied at 400 to 450 pounds pressure with a John Bean Royalette sprayer.

The number of lesions on a leaf is not a true indication of the amount of disease injury, since the location of the lesion appears to be more important in defoliation than the number. A leaf may have 20 lesions and still remain attached to the twig, while another leaf having only one lesion located on the midrib or on the petiole will drop off. Because of this feature, an estimate by observation of the percentage of foliage retention (1) appears to be a more valid indication of disease control than a count of average number of lesions per leaf. Hence the results of these trials are reported in this manner in Table 1.

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[2]Associate Professor of Botany and Plant Pathology.
[3]Trade mark The Upjohn Co., Kalamazoo, Michigan.

FIGURE 1. Appearance of hawthorn trees September 28, 1959. Left -- Treated with 5 ppm Acti-spray. Center -- Untreated control tree. Right -- Treated with 20 ppm Acti-spray.

FIGURE 2. A pair of hawthorn trees located beneath taller elms. Infection had developed until only 65% of the foliage was present before the first application of 5 ppm Acti-spray was made to the right tree only. Appearance September 28, 1958.

One tree which had been treated with Acti-spray at 20 ppm in 1956 was used as an unsprayed control tree from 1957 to 1959. In 1957 leaf blight appeared later than on the other unsprayed control trees but soon built up to almost completely defoliate the tree by October 1.

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MICHIGAN AGRICULTURAL EXPERIMENT STATION, EAST LANSING

CONTROL OF ROOT-KNOT NEMATODE AND APHID ON TOBACCO

Ralph E. Motsinger[1] and O. D. Morgan[2]

Treatment of tobacco soil with the chemical 18133[3] (0, 0-diethyl 0-2 pyrazinyl phosphorothioate) at 10 and 20 pounds per acre of a 5% granular material gave excellent control of the root-knot nematode, Meloidogyne sp., and Myzus sp. of aphid.

Two-gallon crocks were filled with nematode infested soil. The chemical 18133 was applied to the upper 4 inches of the soil at four different times in sets of three crocks. One set was treated 2 weeks, another at 1 week prior to planting, a third at planting time, and a fourth set was applied around established plants 10 days after planting. Both rates were used. There were two plants in each crock. Untreated nematode inoculated controls and untreated uninoculated controls were included.

Previous tests had indicated that the chemical 18133 was systemic and produced small spots on the lower four leaves, as well as some stunting. However, after the appearance of the initial spotting growth was normal and the plants soon caught up with the controls in growth.

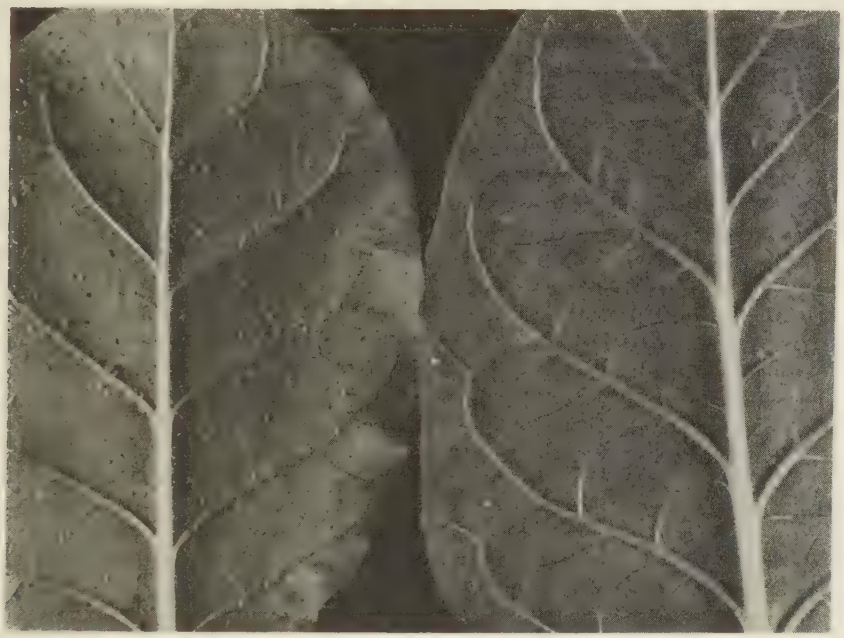

FIGURE 1. Untreated tobacco leaf with aphids at left and leaf from treated plant at right free of aphids.

Because the material was systemic aphids were allowed to build up on the plants to note the effect on these insects (Fig. 1). After 6 weeks aphids were observed only on the untreated controls. All treated plants remained free of both aphids and nematodes after treatment regardless of rate and the time of treatment. Winged aphids that attempted to feed on treated plants died before a new generation could start. Aphids from the controls that found their way to treated plants soon died.

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OBSERVATIONS ON THE EFFECTS OF DECAYING VEGETABLE MATTER ON NEMATODE POPULATIONS[1]

M. T. Hutchinson, J. P. Reed, and D. Pramer[2]

Summary

In a field of barley which had been severely injured by Hoplolaimus tylenchiformis and Pratylenchus pratensis, plant growth in areas where piles of pumpkin had been left to rot appeared to be normal. Tests revealed that parasitic and microphagous nematode populations within these areas were substantially lower than in the rest of the field. There was no apparent correlation between the numbers of predaceous nematodes and nematode-trapping fungi present and the differences in the parasitic and microphagous nematode within the poor and good areas of plant growth.

HISTORY

During October 1958 a barley crop growing in a sandy loam soil at Burlington, New Jersey was observed to have a distinct yellowing of the tip growth, and in some areas plants were dying. An examination of a soil and root sample from this field showed the presence of considerable numbers of Hoplolaimus tylenchiformis, Pratylenchus pratensis, as well as predaceous and microphagous forms, such as:

P. pratensis	-- 250 per gram of root
H. tylenchiformis	-- 500 per gram of root and 700 per pint of soil
Microphagous	-- 500 per pint of soil
Predaceous	-- 550 per pint of soil

In April 1959 the barley stand was very spotty, and large areas had but a few plants. Approximately three dozen areas, several feet in diameter, were distributed evenly across the field in which the barley growth was apparently normal[3]. The grower stated that in 1957 a large crop of pumpkins had been harvested from this field, but, due to a poor market, considerable numbers had been left piled in the field. The location of these piles corresponded to the areas of good growth seen in 1959, as determined by the presence of large numbers of pumpkin seeds.

A good crop of barley was harvested in 1958. Another crop of barley was seeded in early October of 1958, but when it attained a height of about 3 inches yellowing of the tips occurred. Bare spots developed in the field and by the spring of 1959 the situation was as shown in Figure 1.

To determine, if possible, the reasons for the growth difference, soil and root samples were taken from the centers of four of the circular areas of good growth and also from the surrounding areas of poor plant growth. Examinations were made for plant parasitic, predaceous and microphagous nematodes.

In addition, duplicate 5-gram portions from each of the samples were placed on corn meal agar plates and incubated at 20° C for 3 weeks, to determine the presence of nematode-trapping fungi.

RESULTS

Results of the examination for nematodes are shown in Table 1. Both the plant parasitic and microphagous nematode counts for the poor growth areas were considerably higher than where normal plant growth occurred. The predatory nematode counts were divided in this respect.

As for the nematode-trapping fungi, four of eight plates inoculated with soil from the good-growth areas were positive, as were five of the eight plates inoculated with soil from the poor-growth areas. In every case, the fungi were of the Arthrobothrys type. There appeared to be no evidence that these trapping fungi were responsible for nematode population reduction in the areas where the pumpkins had been stacked.

Eighteen months had elapsed, however, between the time the pumpkins had been piled and

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[3]The authors are indebted to Dr. Mack Weed for first observing this phenomenon.

FIGURE 1. Areas of good growth in barley severely injured by Hoplolaimus tylenchiformis and Pratylenchus pratensis, Burlington, N. J., April 6, 1959.

Table 1. Nematodes found inside and outside areas of good-growth in a field of barley, Burlington, New Jersey, April 6, 1959.

Area	Location	Nematodes per pint of soil				Nematodes per gram of roots		
		Hoplolaimus	Pratylenchus	microphagous	predaceous	Hoplolaimus	Pratylenchus	microphagous & predaceous[a]
1	inside	80	0	820	600	80	0	20
	outside	700	0	2800	0	100	0	67
2	inside	400	0	1600	2000	0	0	0
	outside	2920	0	3280	300	275	38	54
3	inside	0	0	420	980	0	0	0
	outside	0	0	1750	5250	0	134	134
4	inside	0	0	1800	1200	0	0	0
	outside	0	0	3760	1740	22	110	268

[a]Types resembling Dorylaimus spp. and Mononchus spp.

when the above data were taken, with no record of conditions in the interim being available. It is reasonable to assume that the large increase in organic matter would have stimulated the population growth of microphagous nematodes, with a resultant increase in at least the predaceous nematode numbers, and possibly the nematode-trapping fungi. These, in turn, would have a reducing effect on the plant parasite population.

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NEMATODES ASSOCIATED WITH RED CLOVER IN ITS SECOND GROWTH YEAR[1]

N. E. Lau and J. P. Reed[2]

Abstract

A nematode survey of 23 second-year red clover fields in eight counties was conducted in the fall of 1957 in order to determine the genera and the population level of nematodes that were associated with red clover. Seventeen genera of stylet-bearing nematodes were found in the soil samples and 11 of these were also found in the root samples. Of the genera known to be parasitic, the following were found: Hoplolaimus, Ditylenchus, Criconemoides, Meloidogyne, Paratylenchus, Pratylenchus, and Tylenchorhynchus. The average number of stylet-bearing nematodes found was 186 per gram of root and 13,897 per pint of soil.

A survey of nematodes from fields of first-year red clover, Trifolium pratense, in New Jersey in 1955 showed seven genera of nematodes to be present in the roots and soil. Rockwood (4) in 1926 reported nematodes as a factor in red clover production in the area around Twin Falls, Idaho. In 1956 Goodey (2) reported five genera of nematodes as being parasitic on T. pratense. These were: Ditylenchus spp., Heterodera sp., Hoplolaimus sp., Meloidogyne sp., and Pratylenchus spp. Twenty-two genera were reported to have been found in the soil around the roots of Trifolium spp. by Jenkins et al. (3). Controlled greenhouse tests by Coursen et al. (1) revealed Paratylenchus projectus and Trichodorus christiei to be parasitic on T. pratense.

Table 1. Nematode study on second-year red clover -- 1957 survey during the period of 9/20 to 11/13. Totals are for incidence of genera of nematodes found in soil and plant samples from 23 fields in eight counties[a].

Nematode genera	Root samples		Soil samples	
	Incidence totals	% of samples each found	Incidence totals	% of samples each found
Aphelenchoides	12	35	10	26
Aphelenchus	56	74	39	69
Boleodorus	-	-	10	13
Criconemoides	-	-	14	22
Ditylenchus	13	30	9	35
Dorylaimidae	-	-	28	65
Helicotylenchus	-	-	11	17
Heterodera	2	4	4	13
Hoplolaimus	52	57	19	39
Meloidogyne	13	17	4	4
Paratylenchus	4	17	78	74
Pratylenchus	48	57	10	22
Rotylenchus	4	9	1	9
Trichodorus	-	-	3	9
Tylenchorhynchus	3	4	19	22
Tylenchus	7	22	25	52
Xiphinema	-	-	7	13
Total	214		273	
Non-stylet-bearing Nematodes	54	74	47	78

[a]Observed species: Heterodera trifolii, Hoplolaimus tylenchiformis, Paratylenchus projectus, P. sp., Pratylenchus scribneri, P. penetrans, P. pratensis, P. n. sp., Tylenchorhynchus claytoni, T. parvus, Rotylenchus robustus, Criconemoides curvatum, C. sp., Xiphinema americanum, Tylenchus costatus, Boleodorus thylactus, Ditylenchus sp., Helicotylenchus sp., Trichodorus sp.

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A nematode survey of second-year red clover fields was conducted in the fall of 1957 to determine the genera and the population level of nematodes associated with red clover. Twenty-three fields in eight counties were sampled for red clover roots and for soil in close proximity to these roots. Root and soil samples were processed separately. The nematodes were isolated and put in known amounts of water, and aliquot portions were taken for nematode counting. Nematodes were taken from each of the processed samples at random and mounted on slides for identification.

Seventeen genera of stylet-bearing nematodes were found in the soil samples and 11 of these were also found in the root samples. In the root samples the incidence of Aphelenchus was greatest, with Hoplolaimus and Pratylenchus following closely. The greatest incidence of nematodes in the soil samples were of the genus Paratylenchus. Twenty-five % of the nematodes found in root samples were non-stylet forms while this figure was 17% for the soil samples. There was considerable injury to the roots by clover root curculios. These insect-feeding injuries were also accompanied by root tissue necrosis. This root decay might be a possible explanation for the high percentage of non-stylet forms found.

Sixty-five % of the genera of stylet-bearing nematodes found in soil samples were also found in root samples (Table 1). This figure could indicate that at least 65% of the stylet-bearing nematodes found in the soil samples were feeding on the roots. However, this figure does not indicate that the other stylet nematodes present were not parasitic or detrimental to red clover plants.

Aphelenchus was found in 74% of the individual root samples with Hoplolaimus and Pratylenchus each being found in 57%. In the individual soil samples, 74% contained Paratylenchus, 69% contained Aphelenchus and 65% contained Dorylaimidae (Table 1).

The percent of the stylet-bearing nematodes per root sample ranged from 40 to 100, with nine samples falling into the 91 to 100% range. Average percent of stylet-bearing nematodes in the root samples for the 23 fields was 79.8. These nematodes per field soil sample ranged from 61 to 100%, with eleven samples falling into the 91 to 100% range. Average for the 23 field soil samples was 85.3%.

Table 2. Nematode study on second-year red clover -- 1957 survey during the period of 9/20 to 11/13. Number of nematodes per gram of roots and per pint of soil from 23 fields in eight counties.

Counties	Fields	Number of stylet-bearing nematodes per gram of root	per pint of soil
Warren	1	177	38,056
	2	186	15,704
	3	16	16,781
	4	383	4,757
	5	102	4,948
	6	78	5,989
	7	32	12,714
Hunterdon	1	7	7,000
	2	42	7,783
	3	170	2,648
Somerset	1	234	4,909
	2	54	3,528
Middlesex	1	30	3,482
	2	126	6,205
	3	1,130	1,477
Monmouth	1	90	48,814
Mercer	1	64	41,665
	2	10	6,178
	3	64	1,514
Ocean	1	80	11,541
Burlington	1	280	4,623
	2	128	14,046
	3	50	5,903
Average		186	13,897

Stylet-bearing nematodes per gram of root ranged in number from 7 to 1130 in the 23 samples, while in the soil samples there was a range of 1477 to 48,814 per pint of soil. The average for the root samples was 186 per gram of root and the average for the soil samples was 13,897 per pint of soil. These data show there was a wide variation in the numbers of stylet-bearing nematodes in the roots and soil from field to field (Table 2).

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CITRUS VARIETIES, HYBRIDS, SPECIES AND RELATIVES EVALUATED FOR RESISTANCE TO THE BURROWING NEMATODE, RADOPHOLUS SIMILIS

H. W. Ford[1], W. A. Feder[2], and P. C. Hutchins[2]

The burrowing nematode, Radopholus similis (Cobb) Thorne, an endoparasite, is reported as the primary cause of the serious citrus disease known as spreading decline (5). An extensive screening program to evaluate all kinds of citrus for resistance or tolerance to attack by the burrowing nematode was started in 1956. The techniques and methods are described in detail elsewhere (1, 2, 3, 4). A partial list of susceptible citrus was published in 1958 (2).

A list of 950 varieties, hybrids, species, and relatives of citrus screened between 1956 and 1960 has been assembled and mimeographed. Of these, 97 kinds were rated of questionable value and will be retested in the soil tanks. Thirty-nine kinds showed sufficient promise to be worthy of greenhouse growth comparison tests in burrowing nematode-infested and noninfested soil. Six kinds grew satisfactorily in greenhouse growth tests and are now being evaluated as rootstocks in infested and noninfested citrus groves. They are Rough lemon A, Rough lemon B, Clone X, Sanguine grosse ronde sweet orange, Pineapple-156 sweet orange, and Carrizo citrange.

Copies of the list can be obtained from the authors.

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TWO VIRUSES THAT INDUCE SYMPTOMS TYPICAL OF "JUNE YELLOWS" IN LETTUCE

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Summary

Two aphid-transmitted yellows type viruses, malva and radish yellows, were isolated consistently either separately or in combination from typical "June yellows"-affected lettuce, Lactuca sativa. In greenhouse tests, these entities have been shown to induce marked interveinal yellowing on several varieties of lettuce. This virus-induced yellowing has been found in high incidence in mature lettuce plantings throughout the growing season.

INTRODUCTION

"June yellows" of lettuce (Lactuca sativa), a general nondescript yellowing of the crop, is common in mature plantings in the Salinas Valley, California. It seems to play an important role in the spring complex of diseases which affect the crop. Also, the disease has been found in certain instances in fields of affected lettuce in the absence of other diseases, such as lettuce mosaic.

"June yellows" has been thought to result from a number of causes, including lettuce mosaic, faulty nutrition, excessive soil salinity, poor root development, unsuitable varieties, unfavorable climatic or soil factors, or combinations of the above (3).

In studies on the malva yellows virus, Costa, Duffus, and Bardin (1) found this entity associated in certain instances with yellowed field lettuce. In additional studies on yellows type viruses isolated from crop and weed hosts in California, in an effort to relate the yellows complex of sugar beet to viruses occurring in other hosts in the region, a distinct aphid-transmitted yellows type virus called radish yellows has been described (2).

Greenhouse tests to determine the host relationships of these two virus entities have involved inoculations of several lettuce varieties. The results of these investigations are described in this report.

GREENHOUSE TESTS

Several varieties of lettuce were inoculated in the greenhouse with a mild isolate of the radish yellows virus and with the malva yellows virus. Colonies of viruliferous green peach aphids were reared on diseased host plants within insectary compartments. Inoculations were made by shaking a large number of insects from the virus source plants onto healthy plants, where they remained for at least 48 hours. After inoculation, the test plants were removed from the insectary, sprayed, and placed in the greenhouse.

Four lettuce varieties, Eiffel Tower Cos, Bibb, Great Lakes, and Prize Head, were inoculated with the viruses; all were susceptible to both entities. The symptoms induced by the two viruses were almost identical except for severity (Fig. 1); the symptoms induced by the malva virus were less severe. Eiffel Tower Cos, Bibb, and Great Lakes varieties reacted similarly to the viruses, exhibiting at first irregular chlorotic blotching, sometimes delimited by the veins, on the older and intermediate leaves. The chlorotic areas tended to coalesce and produce severe interveinal yellowing, especially near the leaf margins and base. Later these leaves turned almost completely chlorotic except for narrow bands along the main veins.

The Prize Head variety reacted with the same type of symptoms; however, instead of chlorosis, there was a reddening of the interveinal areas.

FIELD RECOVERY TESTS

After it became evident that greenhouse infected lettuce plants produced typical "June yellows" symptoms, an attempt was made to determine whether these virus entities were involved in yellowed field lettuce. Recovery tests from field lettuce plants displaying yellowing symptoms were made in the greenhouse. One leaf was selected from each of a number of field plants showing yellowing. These leaves were brought into the greenhouse and their petioles were placed in vials of water. Nonviruliferous green peach aphids, Myzus persicae (Sulz.),

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FIGURE 1. Lettuce leaves of the Bibb variety showing initial symptoms induced by the radish yellows virus. Healthy leaf on the left.

were placed on the leaves for 24 or 48 hours. The aphids were then transferred to radish (Raphanus sativus) for 48 hours and then to healthy sugar beet (Beta vulgaris), cheeseweed (Malva parviflora), and shepherd's purse (Capsella bursa-pastoris) seedlings for 48 hours. A positive reaction on malva seedlings indicated the presence of the malva yellows virus, and a positive reaction on shepherd's purse and sugar beet indicated the presence of the radish yellows virus.

The results of these tests (Table 1) indicate that a high percentage of the plants selected as affected with "June yellows" had malva or radish yellows viruses separately or in combination. The frequency of occurrence of both viruses varied with the location from which the plants were selected. Negative recovery results are believed to be due mainly to technique, although the occurrence of other entities causing similar symptoms is a possibility.

Table 1. Results of virus recovery tests from field lettuce plants showing "June yellows" symptoms.

Field number	Number plants tested	Number of plants from which the indicated viruses were recovered		
		Radish yellows virus	Malva yellows virus	Malva yellows and radish yellows virus
1	10	0	1	6
2	2	0	0	1
3	6	3	0	1
4	10	0	0	9
5	10	0	0	6
6	7	5	0	2
7	10	0	0	10

Isolates of the radish yellows virus from field lettuce plants have differed in severity in their reactions on several host plants. These isolates have not been tested separately as to host range and vector relationships. Although their reactions on several host plants have been similar, some of these entities may represent other yellows type viruses rather than strains of radish yellows virus.

DISCUSSION

The full significance of the two viruses, designated malva yellows virus and radish yellows virus, in the occurrence of the disease of lettuce popularly known as "June yellows" remains to be determined; however, from the evidence already available it seems probable that they may be important causal factors. The widespread occurrence of yellowing observed in certain fields in the Salinas Valley during the past 3 years, sometimes in the absence of extensive mosaic infection, indicates that factors other than mosaic are important in the production of

yellowing in lettuce. The recovery of the viruses of both malva and radish yellows from high percentages of the lettuce plants tested indicates that these viruses, singly or in combination, were present in high percentages of plants in some lettuce fields that showed marked yellowing of the "June yellows" type. The evidence showing that either of these viruses can produce yellowing of lettuce plants under greenhouse conditions further supports the concept that they may be important factors in the "June yellows" problem in the Salinas Valley.

Sources of the two yellows viruses for infection of lettuce in the Salinas Valley probably are abundant and widely distributed. At least six important commercial crop plants grown in the area, as well as a large number of species of weeds, are known to be susceptible to infection with one or both of the yellows type viruses found to be associated with "June yellows" of lettuce.

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STUDIES OF TWO BLUEBERRY STEM DISEASES RECENTLY FOUND IN EASTERN MASSACHUSETTS[1]

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Abstract

Fusicoccum canker and Coryneum canker, two diseases recently found on blueberry in eastern Massachusetts, pose a serious economic threat to the blueberry industry in this area. In Massachusetts the symptoms of Fusicoccum canker vary in some respects from those of this disease in Canada and sporulation occurs over a longer period than reported from Nova Scotia and British Columbia. This indicates that the disease may be more difficult to control in the more southerly areas of its occurrence. Coryneum canker has not been described from any other blueberry-growing area. No effective control is known for either disease.

During the past 3 years stem diseases of the cultivated, highbush blueberry, Vaccinium corymbosum, have become of increasing economic importance in eastern Massachusetts. Fusicoccum canker, a disease known in Canada for over 30 years, was discovered in Massachusetts in 1957 and can now be found in epidemic proportions in some fields. Another disease of somewhat less virulent nature, found in this State in 1956 and called Coryneum canker, occurred in 21 of 25 plantings examined. These diseases, along with Phomopsis canker and gall, may well endanger the economic future of blueberry growing in this area if adequate control measures are not devised. This paper gives the results of studies of Coryneum canker and Fusicoccum canker in eastern Massachusetts.

CORYNEUM CANKER

Coryneum microstictum Berk. & Br. was found for the first time on V. corymbosum by Zuckerman (7), though Cash[3] stated that one collection from V. australe Small in June 1949, which was identified as C. vaccinii Fckl., was probably C. microstictum. The fungus was identified from bushes of the Rubel, Cabot, Pioneer, Berkley, Burlington, Earliblue, Jersey and Pemberton varieties. Further study would probably show that all blueberry varieties under commercial cultivation in this area are affected by this fungus.

C. microstictum, or variants of this species, has been recorded as a parasite on other plant hosts. Beauverie (1) described this fungus as the cause of rose canker. A Corynose twig blight of the American bladder nut, Staphylea trifolia, was described by Davis as caused by C. microstictum var. staphyleae B. H. Davis (3).

The Fungus

The conidia of C. microstictum from acervuli on blueberry stems measure 15.0-24.6 μ x 5.4-8.0 μ. Conidia from culture were of the same size range as those from stems. Jenkins (4) discussed the variability of C. microstictum on rose and concluded that conidia ranged from 10.8-23.0 μ x 4.6-9.0 μ on this host. Mature conidia from blueberry were four-celled. The three terminal cells generally were honey-colored and the basal cell hyaline (Fig. 1D). Conidia were germinated in droplets of sterile distilled water, 1% sucrose solution and on potato-dextrose agar. All of the honey-colored cells were capable of producing germ tubes, and frequently two or three germ tubes were observed emanating from one spore (Fig. 1E). Conidia germinated in 9 to 12 hours at 27° C in the 1% sucrose solution, and by 22 hours had produced germ tubes which were usually 2 to 4 times the length of the conidium, and infrequently as much as 10 times the length of the conidium. Within 22 hours many of the germ tubes had formed septations and were branched. The germinated conidia swelled during this interval and were usually 1.2 times the size of the original ungerminated conidia.

Colonies on potato-dextrose agar attained an average diameter of 1.12 cm after 6 days at 22° C when grown from single conidia. Jenkins (4) stated that C. microstictum var. mali was

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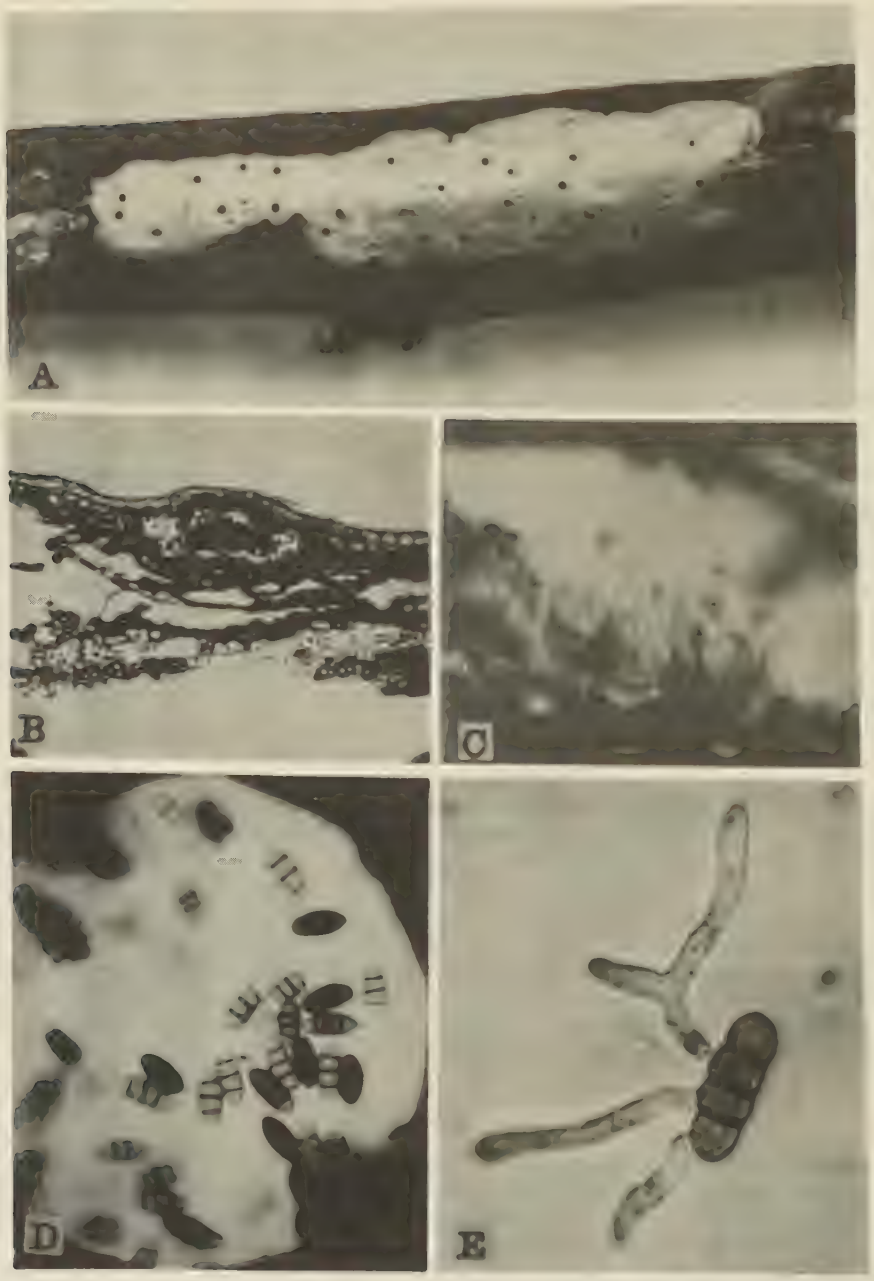

FIGURE 1. A -- Acervuli of Coryneum which have formed in a sunscald area; B -- Cross section of bark showing acervulus which formed immediately below the epidermis; C -- Hymenial layer at the base of the acervulus; D -- Conidia of C. microstictum; E -- Conidium germinated in 1% sucrose solution at 22 hours.

white, C. microstictum from rose was mummy brown to black, and another culture of C. microstictum from rose was tawny olive when grown on potato-dextrose agar for 8 days. Cultures from blueberry were white after growing on potato-dextrose agar for 8 days and after 1 month became brownish black with scattered patches of white. Conidia were formed in approximately 3 months in cultures held at 22°.

The acervuli are erumpent, black, compact, discoid or pulvinate, 159-237 μ in diameter and 58-101 μ high (Fig. 1B). The hyphae ramify through the phellogen-phelloderm layers of

the bark, eventually forming a compact mass which becomes the base of the acervulus (Fig. 1 C). Further development of the acervulus results in a raising of the epidermis. Eventually the epidermis and the cuticle rupture in the form of a triangular opening in the surface of the bark.

Field Studies

In May 1957 a study designed to follow the development of the Coryneum disease syndrome in the field was initiated. The planting in which the experiment was located was poorly drained and the bushes were in low vigor. Twelve branches which contained acervuli of Coryneum, but exhibited no other evidence of decline, were tagged. Frequently the fruiting bodies were associated with sunscalded areas which occurred along one side of the stems (Fig. 1A). These symptoms were considered as representing the incipient stages of the disease. In addition, 11 branches which showed small sunken areas in the bark and contained numerous acervuli within the sunken areas were tagged. This was considered as one of the advanced stages of the disease. The labelled branches were observed at irregular intervals until April 1958, when all branches were removed and carefully examined. Of the 12 branches which bore incipient infections, 5 were dead above the site of infection and 1 was dying. Of the 11 branches which were considered to exhibit symptoms of advanced infection at the beginning of the study, 3 were dead and 4 were dying in April 1958. Three of the dying branches yielded cultures of Phomopsis vaccinii Shear, Stevens & Bain in addition to C. microstictum.

In early March 1959, 12 bushes showing symptoms of incipient infection were tagged. The progress of the disease was observed and by early May 1959 all of the tagged branches were partially or completely girdled by the canker. These results confirm the earlier observations which had been initiated in 1957.

These observations indicate that the area of killed bark extends until the branch is completely girdled. Acervuli develop in abundance and frequently encircle the cankered portions of the branch (Fig. 3A). The portion of the branch beyond the girdle succumbs. The concentric pattern, or bull's-eye, which is associated with Fusicoccum canker is not evident in the Coryneum syndrome. Examination of several hundred Coryneum infections in the field served to confirm this disease syndrome.

Acervuli were frequently found on normal-appearing stems. In two fields where the bushes were in good vigor, the presence of Coryneum had not resulted in a visible decline of the affected branch after 2 years. The most reasonable explanation for this observation is that the fungus is a weak parasite of blueberry, attaining its most serious form on bushes whose vigor has been lowered through other causes.

Acervuli were abundantly produced starting late in February and continuing through the spring, though acervuli which contained viable spores were observed throughout the year. There is no positive evidence to show the manner in which initial infection takes place, but the association of infections with sunscald areas indicates that the pathogen can enter the host through weakened or injured bark tissues (Fig. 1A). The results of inoculation experiments reported in the following section tend to support the observation that only bushes in low vigor are severely affected by the disease.

Infection Studies

Ten bushes of the Jersey variety were inoculated with spores of C. microstictum in May 1957. Inoculum was prepared by making a suspension in distilled water of spores from a pure culture. A single droplet of the suspension was placed on each branch, then the branch was wounded twice by pricking the bark within the area covered by the droplet with a sterile needle. In February 1958, eight bushes of the Pioneer variety were treated with atomized spore suspensions of C. microstictum in the greenhouse. Four of the branches were then wounded by pricking the bark several times with a needle and four were not wounded. In addition, sterile distilled water was atomized on one stem of each of four bushes and the stems wounded as before. In April 1958, 20 bushes of the Pioneer variety located in the screenhouse were inoculated in the same manner. Spore suspensions were atomized on one branch of each of 16 bushes and 12 of these branches were wounded by pricking with a sterile pin. Sterile distilled water was atomized on one branch of each of four bushes and the branches wounded as before. Inoculation trials were repeated in March 1959 on 20 bushes of the Rubel variety. In these tests a spore mass taken directly from an acervulus on a naturally infected branch was placed under a flap of bark cut into the stem of each of 15 bushes. Spore masses also were placed on unwounded stems of each of five bushes.

In all of the preceding trials except five, inoculated branches were covered by plastic bags. The plastic bags, which served to maintain high humidity in the area of the wound, were removed 48 hours following inoculation.

The results of these experiments are given in Table 1. The introduction of spores into wounded tissues, or the placing of inoculum in contact with wounded tissues under conditions of high humidity, resulted in infection in 20 cases out of a total of 49 trials. Acervuli formed near the site of inoculation but the infections were generally walled off. However, in one case a well-defined, girdling canker was formed following inoculation. The resistance of the host to the spread of the fungus within its tissues once infection had become established indicates that C. microstictum is a weak pathogen on vigorous bushes. Infection through unwounded host tissue did not occur in any of the 13 trials. These results suggest that infection may take place only through damaged host tissues.

Table 1. Inoculation trials of pathogenicity of C. microstictum to the highbush blueberry.

Date inoculated	Number plants inoculated	Treatment	Variety	Location	Results: positive	Results: negative
May, 1957	10	Spore suspension placed on stem, stem wounded	Jersey	field	5	5
Feb., 1958	4	Spore suspension atomized on stem, stem wounded	Pioneer	greenhouse	-	4
	4	Spore suspension atomized on stem, unwounded stem	Pioneer	greenhouse	-	4
	4	Sterile distilled water atomized on wounded stem	Pioneer	greenhouse	-	4
Apr., 1958	12	Spore suspension atomized on stem, stem wounded	Pioneer	screenhouse	6	6
	4	Spore suspension atomized on unwounded stem	Pioneer	screenhouse	-	4
	4	Sterile distilled water atomized on wounded stem	Pioneer	screenhouse	-	4
Mar., 1959	15	Spore mass placed under bark flap on stem	Rubel	field	9	6
	5	Spore mass placed on unwounded stem	Rubel	field	-	5

Control

To date no effective control measures have been evolved. Three applications of ferbam, ziram or phenyl mercury lactate in the spring of 1958 failed to give control. In the fall of 1959 a series of pruning experiments aimed at controlling the disease were initiated; however it may be several years before the results of these treatments can be evaluated.

FUSICOCCUM CANKER

Fusicoccum canker, caused by the fungus Fusicoccum putrefaciens Shear, was found in Massachusetts for the first time in 1957 (6). A severe outbreak occurred in several plantings in the State in 1958, and in 1959 the disease appears to be more widespread. To date, the disease has been found in eight plantings and has been identified on bushes of the Rubel, Jersey, Cabot and Pioneer varieties.

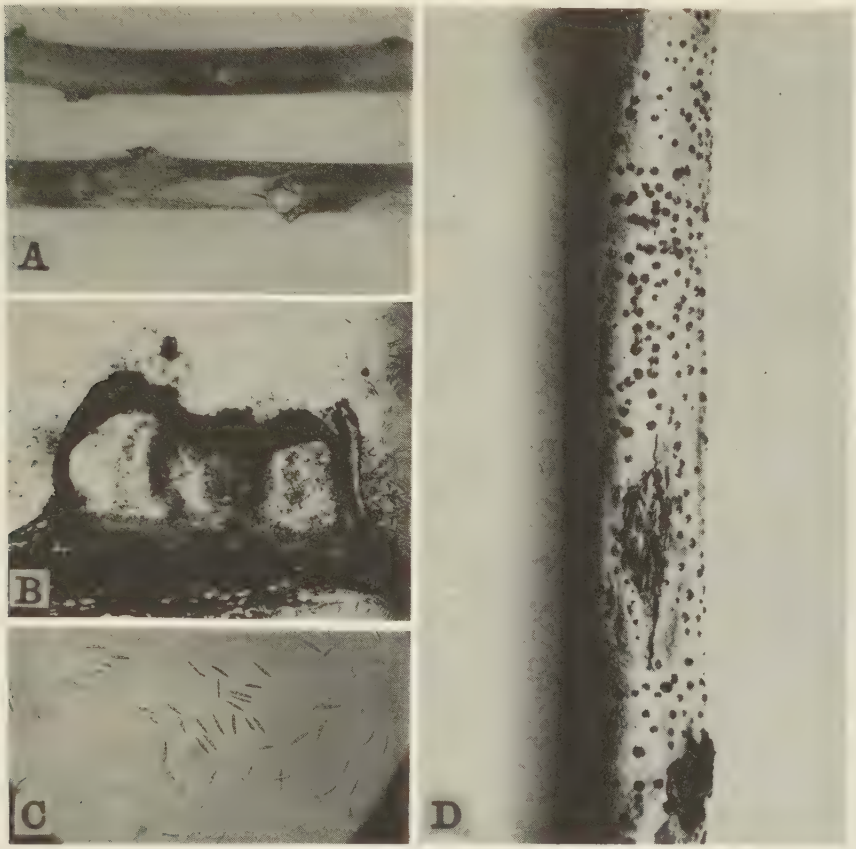

FIGURE 2. A -- Initial dark red lesions surrounding bud, upper twig, and advanced, girdling canker, lower twig. Note numerous erumpent pycnidia in central portion of canker; B -- Cross section of chambered pycnidium; C -- Conidia of F. putrefaciens; D -- Large erumpent pycnidia on severely cankered stem.

FIGURE 3. A -- Abundant acervuli on a branch heavily infected with C. microstictum; B -- The "bull's-eye" pattern, a distinctive symptom of Fusicoccum canker.

Fusicoccum canker was observed in Quebec in 1931. In 1958 McKeen (5) described the disease syndrome as it occurs in British Columbia, and reported on investigations which established the pathogenicity of Fusicoccum on the cultivated highbush blueberry. Concurrently, Creelman (2) published a similar paper which related to investigations of this disease in Nova Scotia.

Field Symptoms

Creelman (2) and McKeen (5) report that infection of blueberry stems by F. putrefaciens apparently takes place in the fall. The fungus overwinters as mycelium on the living, woody portions of the plants. Frequently cankers form in the region of the root crown or at the base of the branches. In Massachusetts initial lesions appear on 1-year-old branches during late winter and early spring. The lesions, which are first dark-red and circular to elliptical, quickly enlarge in the spring and completely girdle the stem (Fig. 2A). The wood beneath the lesions exhibits a brownish discoloration. Creelman (2) states that in Nova Scotia pycnidia appear in dead bark in cankers for about 5 weeks beginning in late July. In southeastern Massachusetts sporulating pycnidia were found in abundance from the first of March through mid-July. McKeen (5) reported that pycnidial initials form in early March in British Columbia.

Fusicoccum canker may appear in a "bull's-eye" pattern around the locus of infection (Fig. 3B). This symptom is caused by alternating periods of active growth and inactivity of the pathogen within the host tissue. Each period of active growth is evidenced by a band of differentially colored host tissue encircling and contiguous to the area previously invaded by the fungus. This characteristic, and the distinctive appearance of the erumpent pycnidia, facilitate field identification of the disease (Fig. 2D). In 1958 and 1959 the "bull's-eye" symptom occurred on about 20% of the diseased specimens collected during March and April.

Creelman (2) and McKeen (5) in their separate description of the field symptoms of the disease do not report on finding the "bull's-eye" symptom, and it may be that this does not occur in Nova Scotia and British Columbia.

Infected stems begin wilting and dying in May, and wilting and dying of branches continue throughout the summer. Stems which had exhibited small lesions in February 1959 were girdled by April 1959, and killed by June 1959. In this case the time interval between the formation of visible symptoms and death was about 18 weeks. The rate of development of symptoms is probably dependent upon climatic conditions; therefore, this will vary from year to year.

The disease generally kills one to three branches on a bush, but occasionally the entire plant will be killed. In one field in Eastern Massachusetts about 40% of the plants were affected. The overall effect is a reduction in yield of the planting, and a decline in vigor of the individual bush.

The Fungus

The apothecial stage of the causal organism, Godronia cassandrae Peck is found on old prunings and pruning stubs (7). Fusicoccum putrefaciens Shear, the pycnidial stage of the fungus, fruits abundantly within the cankers formed on the branches. The pycnidia are subglobose to pyriform, brownish, sessile or subsessile, simple or irregularly chambered 160-400 μ in diameter, others with large chambers 400-450 μ in diameter (Fig. 2B). Spores are hyaline or faintly yellowish in mass, elliptic to fusiform, continuous or pseudoseptate, and 8.0-19.0μ x 2.0-3.5μ (Fig. 2C).

Isolates on potato-dextrose agar from blueberry taken from the brownish discolored bark areas underlying the incipient cankers yielded yellowish, raised colonies similar in appearance to those which were isolated from cranberries, Vaccinium macrocarpon. On cranberries F. putrefaciens is the cause of a late storage rot. McKeen (5) in a comparison of Fusicoccum from cranberry and blueberry described differences in morphology and pathogenicity. Conidia from blueberry in Massachusetts are larger than those reported from blueberry in British Columbia and similar in size to those from cranberry in Massachusetts. Further comparison of cranberry isolates with blueberry isolates from all locations where the disease is found should lead to a clear understanding of McKeen's findings and those reported herein.

Field Observations and Inoculation Trials

The development of Fusicoccum canker in the field was followed by means of tagging 12 bushes which showed small dark red, incipient lesions surrounding buds. In early March 1959, when the study was initiated, these lesions did not contain fruiting bodies. By mid-April fruiting bodies were evident in lesions on eight of the bushes. Observations on May 4 showed that all of the cankers contained well-developed, erumpent pycnidia. Several branches on all 12 bushes had been completely girdled, although at this time the portions of the branches above the cankers had not died.

In April 1958 seven bushes of the Pioneer variety located in a screenhouse were inoculated with conidia or mycelial transplants of F. putrefaciens. In March 1959 6 Rubel bushes in the field were inoculated with conidia. Inoculation techniques were the same as those outlined under the section on Coryneum canker. Typical Fusicoccum girdling cankers developed on four of the bushes inoculated in 1958 and on three of the bushes inoculated in 1959. McKeen (5) reported that infection resulted from inoculation with conidial and ascospore isolates made in November and December, but not from inoculations made in June and July.

Control

Creelman (2) reported that a series of chemical treatments applied in the spring of 1952 failed to prevent canker formation or eradicate existing infections. In the spring of 1958 ziram, ferbam and phenyl mercury lactate were applied in control experiments in Massachusetts. The results were similar to those of Creelman. McKeen (5) experienced a similar failure in British Columbia. He suggests that fungicides applied in the fall should give control. To date, there has been no published report of successful chemical control.

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FUNGI COLLECTED FROM BLUEBERRY STEMS IN MASSACHUSETTS[1]

Bert M. Zuckerman[2]

During the course of a study of fungi which occur on stems of the cultivated highbush blueberry, Vaccinium corymbosum, several fungi hitherto unreported from living stems of this host were collected and identified. These were Rhabdospora oxycocci Shear and an Epicoccum sp. from Rubel, Pezizella lythri (Desm.) Shear & Dodge from Jersey, a Macrophoma sp. from Cabot and Berkeley and Karschia lignyota (Fr.) Sacc. from Pemberton. The last was found only on exfoliating bark.

A Septoria sp. collected from Rubel stems was tentatively identified as S. vaccinii E. & E. This fungus has been reported from the leaves of Vaccinium, but not from the stems. An Alternaria from Rubel and Dixi bushes was identified as A. tenuissima (Fr.) Wilts. Simmons states that in his opinion there are several species in the A. tenuis-A. tenuissima group, therefore this identification should be considered as tentative pending further taxonomic study[3]. An Alternaria has been reported as a secondary invader in leaf and twig blight of V. corymbosum but the fungus was not identified to species[4].

Dead stems were examined on many occasions. Fungi found which were previously unreported from dead stems of this host were Pleospora obtusa (Fckl.) Hoehn. and two unidentified species of Cladosporium. In addition, Strasseria oxycocci Shear was collected from a recently killed twig tip.

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[3]The author wishes to thank Dr. L. E. Wehmeyer for his identification of Pleospora obtusa and Karschia lignyota and Dr. E. G. Simmons for his opinion as to the identity of the Alternaria reported herein.

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CORM AND SOIL TREATMENT FOR THE CONTROL OF BACTERIAL SCAB OF GLADIOLUS

Lester P. Nichols[1]

Bacterial scab, caused by Pseudomonas marginata (McCull.) Stapp, is one of the most common diseases of gladiolus in Pennsylvania. Lesions on the corms usually do not reduce the production of flowers, although the neck rot phase of the disease may cause serious reductions in yield.

In 1954 Young (3) reported control of scab by corm treatments of fungicide-insecticide mixtures. Forsberg (1) achieved control of this disease by corm treatment using Emmi (N-ethylmercuri-1,2,3,6-tetrahydro-3,6,-endomethano-3,4,5,6,7,7-hexachlorophthalimide), a liquid mercury fungicide, in combination with a soil treatment of either aldrin, lindane, or heptachlor at planting time. He later reported (2) the control of scab by the addition of aldrin or heptachlor to the soil at planting time with no preplanting corm treatment.

The standard recommendation for the control of scab and other gladiolus diseases in Pennsylvania has been a corm dip at planting time of calomel (mercurous chloride) or New Improved Ceresan (5% ethyl mercury phosphate). Control of scab and other diseases has not always been satisfactory. In addition, the use of these materials has often resulted in delayed emergence of the shoots and a delay in flowering.

In 1958 a test for control of bacterial scab was carried out in a commercial gladiolus field in Somerset County, Pennsylvania. Corm treatments of Delsan A-D (60% thiram, 15% dieldrin), Emmi, and corms treated with Emmi planted in soil treated with heptachlor, were compared with the standard corm treatments of calomel and New Improved Ceresan, and an untreated check. Due to the low incidence of disease, no information on disease control was obtained. However, the treatments containing mercury delayed the date of emergence of the shoots and the date of flowering by almost 2 weeks.

In 1959 the test was repeated. The treatments were as follows:

1. Delsan A-D, dusted on at the rate of 2 level tablespoons per 100 corms.
2. Emmi, 1 cup per 25 gallons of water, 2-hour soak of corms.
3. Emmi, corm soak with treatment number 2; corms planted in rows treated with 5% granular heptachlor, 63.6 grams sprinkled per 100 feet of row.
4. Calogreen (90% mercurous chloride), 2 level tablespoons per gallon of water; corms dipped until wet.
5. New Improved Ceresan, 1 level tablespoon per gallon of water; 15 minute soak.
6. Untreated check.

Two hundred corms of each of the following varieties were used for each treatment: Bridal Orchid, Mother Fisher, and Washington. Before treatment 100 corms of each variety for each treatment were husked. None of the husked corms showed any evidence of scab infection. After treatment the corms were planted in rows, one treatment per row, 100 husked and 100 unhusked of each of the three varieties per row. The corms were planted on May 7, 1959.

Table 1. Effect of corm treatment on emergence, corm weight, and control of scab on gladiolus.

Treatment	Emergence (600 corms planted)	Average weight of new corms (in ounces)	% scab on new corms
Delsan A-D	578	1.42	36.0
Emmi-heptachlor	485	1.47	46.0
Emmi	537	1.34	69.5
Calomel	568	1.16	79.0
New Improved Ceresan	583	1.24	66.0
No treatment	575	1.22	68.5

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RESULTS

Effects on Emergence: Emergence counts made on June 12 are summarized in Table 1. The Emmi corm treatment planted in heptachlor treated soil had a delaying effect on the emergence of the plants.

Effects on Flower Production: There was no outstanding difference in total flower production with any of the treatments. The grower observed a delay of flowering of up to 2 weeks with all the treatments containing mercury.

Effects on Corm Weight at Harvest: The corms were harvested on October 6 and the new corms were counted and weighed. Average weight per corm in ounces is shown in Table 1. Plants from the New Improved Ceresan treatment produced the largest number of corms while those from the calomel treatment produced the smallest number. Average corm size was 1.47 ounces in the Emmi-heptachlor treatment and 1.42 ounces in the Delsan A-D treatment. The lightest corms were in the calomel treatment at 1.16 ounces per corm.

Effects on Control of Scab: On November 20, 30 of the new corms of each variety in each treatment were husked and checked for the presence of scab. It was noted that a corm was scabby or was free of scab. The results of this count are shown in Table 1 as the percentage of scab on new corms. Corms treated with Delsan A-D produced corms that had 43% less scab than those produced by corms treated with calomel. Corms treated with Emmi and planted in heptachlor treated soil produced new corms that had 33% less scab than those produced by corms treated with calomel. None of the other treatments were as effective in controlling scab as the Delsan A-D and the Emmi plus heptachlor treatments.

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EVALUATION OF COTTON STRAINS AND PROGENIES FOR RESISTANCE TO VERTICILLIUM WILT[1]

Alfred B. Wiles[2]

Summary

By use of a seedling-inoculation technique, strains of Upland cotton highly tolerant or resistant to Verticillium albo-atrum in the seedling stage were developed by screening and selection. This resistance was found in Fusarium-resistant strains of cotton listed as Alabama Hybrids 56-4 M, 257-202, and 81-14. Under field conditions the resistance to Verticillium wilt in these strains tended to be less pronounced in the mature plant than in the seedling stage. Strains of G. arboreum, G. barbadense, and G. herbaceum were highly resistant to the causal organism in both greenhouse and field tests, while strains of G. harknessii and G. klotzchianum were not.

INTRODUCTION

Verticillium wilt of Upland cotton (Gossypium hirsutum), caused by Verticillium albo-atrum Reinke & Berth., was first reported by Carpenter (1) in 1914; four years later (2) he reported that he was able to induce the same disease in cotton with isolates from okra. Sherbakoff (6) reported presence of the disease in Tennessee-grown cotton in 1928, and Miles and Persons (4) identified the same disease in Mississippi-grown cotton in 1930. At present Verticillium wilt occurs to some extent in most cotton-growing areas of the world. In the United States it is found throughout much of the Mississippi Valley and more particularly in the irrigated areas of the Southwest. In some years (1952, 1953, 1954) the disease was barely evident in Mississippi; in others mild outbreaks occurred (1951); and in still others the disease has been very destructive (1950). If the disease is not severe a moderately tolerant variety will perform satisfactorily in the field; however, when the disease is severe none of the common commercial varieties is sufficiently tolerant to prevent considerable loss from Verticillium wilt. Because of the nature of the disease, as well as of the crop, the development of resistant or tolerant varieties is considered the most practical approach to control. The objects of the present study were to determine whether a usable level of resistance existed in Upland cotton and whether possible sources of resistance existed in other species of cotton.

The susceptibility to Verticillium wilt of most Upland cottons and the resistance or tolerance shown by various cottons of Gossypium barbadense had already been noted by previous workers (5). These findings were primarily obtained by field tests. The tendency of cotton plants to escape infection, the sporadic occurrence of the disease in a given field, and the season-to-season variability in severity in Mississippi make it necessary to employ further means of testing and evaluation. The use of seedling inoculation was found satisfactory for this purpose in other crops (7) and Drummond (3) used a type of artificial inoculation for cotton. The method reported herein was used on cotton under greenhouse conditions in Mississippi during the fall, winter, and early spring, when sufficiently cool temperatures could be maintained to obtain infection.

Since both Fusarium wilt (Fusarium oxysporum Schlecht f. vasinfectum (Atk.) Snyder & Hansen) and Verticillium wilt occur on cotton in Mississippi it is very desirable to obtain resistance to both diseases within a single strain or variety. Therefore, the Fusarium-resistant varieties received the most intensive screening for resistance to Verticillium wilt.

INOCULATION TECHNIQUE AND EVALUATION

A report (8) was given on the testing technique used. It consisted of planting acid-delinted cotton seed in steam-sterilized sandy loam in seed flats. The inoculum was prepared by growing the fungus in a liquid nutrient and then mixing it in a Waring Blendor. Sufficient plain agar was added to give the inoculum a thick consistency. When the seedlings were in the four-

[1]Journal paper (New Series) No. 845, Mississippi Agricultural Experiment Station. Cooperative investigations of the Crops Research Division, Agricultural Research Service, United States Department of Agriculture, and the Mississippi Agricultural Experiment Station.

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Table 1. Reaction of cotton seedlings to Verticillium albo-atrum.

Species, and variety or strain	Number of plants evaluated	% of plants surviving 30 days after inoculation
Amphidiploids (n = 26):		
Gossypium hirsutum		
1. Acala (Strain W 29-16)	34	9.5
2. Alabama Hybrid 56-4 M	39	41.0
3. Alabama Hybrid 81-14	35	44.8
4. Alabama Hybrid 257-202	33	42.0
5. Ambassador	36	3.8
6. Delta Pine 15	36	7.7
7. Empire (Strain 8-0-8)	1130	13.9
8. Empire (Strain 8-6-1)	939	13.8
9. Empire (Strain P-47)	34	15.6
10. Hartsville (Strain W-1)	38	25.0
Gossypium barbadense		
1. Sea Island (Seabury)	44	82.5
Diploids (n = 13):		
Gossypium arboreum		
1. Strain 1	37	94.1
2. Strain 2	37	76.5
3. Strain 3	37	82.3
4. Strain 4	27	85.7
5. Strain 5	24	95.8
Gossypium harknessii		
1. Strain 1	46	8.7
Gossypium herbaceum		
1. Strain 1	64	90.6
2. Strain 2	62	35.5
Gossypium klotzchianum		
1. Strain 1	32	8.3
2. Strain 2	37	5.9

leaf stage (about 20 days after planting) they were inoculated by lifting them from the soil with a spatula and dipping the roots in the inoculum. The plants were then reset in the same soil. If environmental conditions favored disease development wilting of the young plants occurred within 7 to 8 days after inoculation. A discoloration of the vascular tissue similar to that found under field conditions was evident in these plants and the wilt organism was readily isolated from the plants. Many seedlings died within 30 days after inoculation and final evaluations were made by using living (resistant or tolerant) and dead (susceptible) classifications.

EXPERIMENTAL RESULTS

Greenhouse results: A preliminary report (9) was given on the reaction of cotton varieties to Verticillium wilt when the seedling-inoculation technique was used. The results obtained from screening selected varieties and strains of *G. hirsutum* and other *Gossypium* species by using this method were recorded (Table 1). From Alabama Hybrids 56-4 M, 81-14, 257-202, Empire 8-0-8 and 8-6-1, and Hartsville W-1 surviving individual plants were saved and self-pollinated. The results of evaluating the seedling progenies of certain of these surviving plants are shown (Table 2). Some of the selections produced progenies that gave a resistant reaction (Fig. 1) and others did not. In subsequent screening tests additional selections were made from these resistant lines and progeny evaluations have indicated that a high level of resistance has been maintained in the seedling stage by these strains. The high degrees of resistance to Verticillium wilt of *G. barbadense*, *G. arboreum*, and *G. herbaceum* in the seedling stage are shown (Table 1).

Field Results: After sufficient seed were obtained a field evaluation of the strains was made. From these tests it was found that where Verticillium wilt occurred naturally the resistance to Verticillium wilt exhibited in the seedling stage of *G. hirsutum* was less pronounced

Table 2. Reaction to Verticillium albo-atrum of progenies of certain cotton plants surviving seedling inoculation with the fungus.

Strain and plant number		Number of progenies evaluated	% surviving 30 days after inoculation
Ala. Hybrid (81-14):	4-1	26	37.5
	4-2	22	0.0
	4-3	25	0.0
	4-4	24	71.4
	4-5	27	81.3
	9-1	24	35.7
	9-2	23	76.9
	9-3	26	93.8
	9-4	23	76.9
	9-5	26	81.3
Hartsville (W-1):	17-1	29	5.3
	17-2	17	0.0
	17-3	10	10.0
	17-4	15	0.0
	17-5	17	0.0
	17-6	13	23.1
Empire 8-0-8:	38-1	28	0.0
	38-2	30	10.0
	38-3	27	37.0
	38-4	26	20.0
	38-5	25	0.0
	38-6	30	0.0
	38-7	27	0.0
	38-8	29	15.8
	38-9	28	0.0
Empire 8-6-1:	61-1	30	10.0
	61-2	30	0.0
	61-3	29	15.0
	61-4	30	15.0
	61-5	28	0.0
	61-6	28	5.3
	61-7	27	0.0
	61-8	26	25.0
	61-9	23	15.8
	61-10	23	15.4
Ala. Hybrid (56-4 M):	89-1	26	62.5
	89-2	24	37.1
	89-3	28	
	89-4	23	
	89-5	28	
Ala. Hybrid (257-202):			
	92-1	29	15.8
	92-2	32	13.2
	92-3	28	0.0
	92-4	27	11.8
	92-5	21	36.4
	92-6	28	3.6
	92-7	24	42.9
	92-8	23	53.8
	92-9	22	56.7

in mature plants. Resistance to the disease was exhibited by mature plants of resistant strains developed by the seedling inoculation process, but not at a level comparable with the resistance shown in the seedling stage. Conversely, Delta Pine 15 was very susceptible in the seedling test, but under field conditions it was moderately resistant. G. barbadense, G. arboreum, and G. herbaceum were resistant to the fungus under field conditions.

FIGURE 1. Reaction of the progenies of three cotton plants which survived seedling inoculation with Verticillium albo-atrum. Numbers 228 and 118 were classified as susceptible and number 54 was resistant.

A high percentage of the F_1 progenies of crosses between G. barbadense and a susceptible G. hirsutum were resistant in the seedling stage. A crossing program involving this source of resistance to Verticillium wilt was initiated.

DISCUSSION AND CONCLUSIONS

In previously reported work, testing for resistance to Verticillium wilt of cotton was usually conducted in a field plot where previous plantings had demonstrated a high degree of wilt infestation. This was the only available method when no dependable technique for creating artificial epiphytotics was known. It appeared at the outset that more rapid progress could be expected when an effective seedling-evaluation technique was used in conjunction with field testing and other established breeding techniques.

The results of the study suggested that the expression of symptoms of Verticillium wilt in mature plants of G. hirsutum under disease conditions in the field was not conditioned entirely by the genetic resistance or susceptibility of the host. It is thought that host nutrition as well as certain environmental factors may possibly influence symptoms of disease expression. In connection with the resistance exhibited by other species of Gossypium it should be noted that members of these species are different from G. hirsutum in almost all aspects of growth habit, maturity rate, and genetic background. It was determined that there exists a high resistance to Verticillium albo-atrum within G. hirsutum in the seedling stage of certain varieties. This resistance is less pronounced in the mature plant and is apparently subject to influences other than host resistance. It was concluded that a more stable source of resistance than that in G. hirsutum was desirable and that such resistance was available in G. barbadense.

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FOMES ANNOSUS ROOT ROT OF LOBLOLLY PINE

P. C. Lightle

This paper records the first known damage to loblolly pine (Pinus taeda) by the fungus Fomes annosus (Fr.) Cke. The organism causes serious root and butt rot losses in European coniferous plantations. In North America it has been reported in both hardwood and coniferous forests. The causal fungus is listed in the "Index of Plant Diseases in the United States"[1] as occurring on various pine species but not on loblolly pine. Though Seymour's Host Index[2] records its presence on loblolly, a search of the literature disclosed no report of its causing damage to this species.

The first occurrence noted was in a small 21-year-old plantation of loblolly pine on the Biloxi Ranger District of the DeSoto National Forest, near Gulfport, Mississippi. The plantation had been first thinned at age 15 years, and rethinned 4 years later. A few months after the latter thinning a few dead trees were noticed. The cause of mortality was thought by National Forest personnel to be Ips beetles, and the dead trees were felled and sprayed with benzene hexachloride. The following year additional trees died and the area was again treated for Ips. When mortality recurred in the spring of 1959, forest personnel suspected that the insects were secondary. Pathologists, investigating, found symptoms typical of Fomes annosus, that is, dead cambium at the root collar, dead pitch-infiltrated roots, and characteristic buff and russet coloration of separated wood and bark.

Nineteen of 27 cultures from decayed roots and stump wood were F. annosus. Identification was confirmed by Ross W. Davidson of the U. S. Forest Disease Laboratory, Beltsville, Maryland.

Examination of the area in July 1959 disclosed a number of fruiting bodies of the fungus. Most of them were 1 to 3 feet away from the stumps of cut trees, on lateral roots, and completely covered by duff. A few were at ground-line on the stumps.

In August 1959, F. annosus was isolated from the extensively decayed roots of two dying mature loblolly pines in a natural stand in central Alabama. Thus we now know of two widely separated loblolly pine stands currently suffering mortality from this pathogen.

SOUTHERN FOREST EXPERIMENT STATION, FOREST SERVICE,
UNITED STATES DEPARTMENT OF AGRICULTURE

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OBSERVATIONS ON PUSTULAR SPOT ON PEACHES

W. R. Wright, M. A. Smith, G. B. Ramsey, and L. Beraha[1]

In September of 1957 shipments of Colorado peaches arriving on the Chicago market showed pustular spot, caused by Clasterosporium carpophilum (Lév.) Aderh. Other than the note by Rose[2] that pustular spot may develop in transit at temperatures above 50° F, information is lacking on the effect of transit and storage temperatures on the development of this disease.

A series of measurements was made of 33 representative, isolated pustular spots on peaches taken from a truck shipment originating in Colorado. Half of the specimens were held at 72° F and the remainder at 42°. Increment increases in diameter measured at intervals of 2, 3, and 7 days averaged 1.6 mm, 1.31 mm, and 1.46 mm at 72° F; and 0.45, 0.34, and 0.16 mm respectively for those specimens held at 42°.

Forty Colorado peaches were wounded by a shoulder puncture with a No. 20 cork from which projected six pinpoints. The fruit was then rolled in a water spore suspension of the pustular spot organism. Ten peaches each were placed at 80°, 75°, 70°, and 40° F. Fruits punctured and rolled in sterile distilled water served as controls. Examination after 3 days revealed initial infections characterized by small water-soaked specks scattered over the shoulder and blossom-end areas of the fruit held at 75°. None of the peaches held at the other temperatures showed infections at this time. At the end of 1 week many lesions were present on the peaches held at 70° and 75°. Early infections were also observed on the peaches held at 80°. No infections were visible on the inoculated peaches held at 40° (Fig. 1). Control fruits remained uninfected. Wounded and non-wounded areas of inoculated fruits appeared equally susceptible.

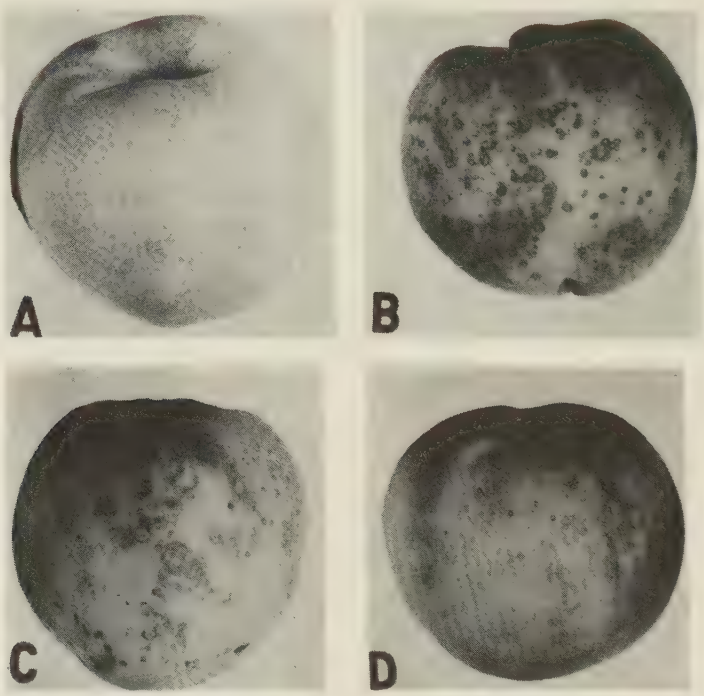

FIGURE 1. One-week progress of pustular spot on inoculated peaches. A -- 40° F; B -- 70°; C -- 75°; D -- 80°.

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[2]Rose, D. H. 1924. Diseases of Stone Fruits on the Market. U. S. Dept. Agr. Farmers' Bull. 1435.

After an 8-day holding period, peaches from the above lot held at 80° showed lesions ranging from 0.5 to 6 mm, averaging 2.27 mm in diameter. Lesions on fruit held at 75° ranged from 1 to 10 mm, averaging 3.85 mm in diameter. Infections on fruit held at 70° ranged from 1 to 7 mm in diameter, averaging 4.07 mm. Peaches held at 40° remained uninfected.

After 15 days peaches held at 40° F were still free of visible infection. Six of these fruits were transferred to 54°. Examination 24 hours later revealed tiny water-soaked spots on them. Within 5 days all of the fruits were heavily infected with typical pustular spot. Some lesions measured 4 mm in diameter. Many of the lesions had coalesced.

Twenty days after start of the experiment pustular spot was noted on three of the four remaining peaches being held at 40° F. Lesions were few and scattered, with a maximum size of 4 mm.

In another inoculation series, fruit was held at 80° F, room temperature (59° to 75°, average 68°), 54°, and 40° F. Initial infections were visible on fruit after 4 days at room temperature, 5 days at 80°, and 6 days at 54°. Peaches held at 40° remained free of visible infection after 7 days.

Spore germination and incipient infections are believed to have taken place on fruits held at 40° F (this was indicated by the appearance of infection 24 hours following transfer from 40° to 54°), whereas fruits inoculated and then incubated at 54° did not show visible infection for 6 days. The eventual appearance of pustular spot on inoculated fruit held continuously at 40° also indicated establishment of infection at this temperature.

Table 1. Clasterosporium spore germination at various temperatures.

	80° F	75°	55°	40°
Incubation period	16 hours	17 hours	18 hours	19 hours
Number of spores observed	3152	1445	1620	1273
Percentage germination	97	88.8	72	36.2
Percentage uni-polar germination	15.8	33.2	28.5	46.7
Percentage bi-polar germination	47.7	26.3	48.5	17.7
Percentage other[a]	36.4	40.4	22.8	35.4
Maximum germ-tube length (μ)	550	700	350	160
Average germ-tube length (μ)[b]	307	520	176	53

[a] Uni- or bi-polar plus one or more germinating interior cells.
[b] Only the maximum germ-tube length per field was recorded.

An experiment was initiated to test the germinability of Clasterosporium spores at various temperatures. Spores were seeded on water-agar plates. Three plates each were placed at temperatures of 80°, 75°, 55°, and 40° F. The experiment was repeated three times and data are summarized in Table 1.

Observations during the progress of the germination tests demonstrated that Clasterosporium spores can germinate within 2 1/2 hours at temperatures of 75° and 80° F, in 3 hours at 55°, and in 8 hours at 40°.

MARKET QUALITY RESEARCH DIVISION, AGRICULTURAL MARKETING SERVICE, UNITED STATES DEPARTMENT OF AGRICULTURE

INFECTIOUS VARIEGATION OF CITRUS FOUND IN FLORIDA

Theodore J. Grant and Paul F. Smith[1]

Summary

Two grapefruit trees with crinkled and irregular white to yellowish blotches on the foliage of some branches were found at Cherry Lake, Florida following the severe, cold winter of 1957. Buds and leaf pieces from these transmitted the causal agent of the disease to several different citrus varieties. Pruning and defoliation were used to stimulate symptom expression. In general the symptoms produced were characteristic of those described for infectious variegation. The occurrence of crinkle leaf and veinlet banding in some inoculated plants indicated the presence of additional strains of the psorosis virus complex.

Infectious variegation was first described by Petri (4) in 1931. He found the disease on sour orange, but not on the sweet orange or mandarin plants, in a citrus nursery in Sicily. He did not obtain transmission by inoculations with juice of the variegated foliage or by grafts to healthy plants. However, his cytological studies indicated the variegation was more like that of a mosaic, caused by a virus, than like a chimaera associated with a bud mutation, thus he considered the disease to be infectious. His pictures and description of diseased foliage are similar to those reported by Fawcett and Klotz (2) in 1939. The affected leaves had areas of variable size that lacked green color and were white to yellowish white. These areas were not arranged in any regular pattern. In some leaves these areas were mostly on one half of the leaf blade as divided by the midrib. The causal agent was transmitted by buds from an infected lemon tree to sour orange plants by Fawcett and Klotz (2). In addition to the chlorotic areas, in the small rapidly-growing leaves Fawcett and Klotz also observed flecking symptoms characteristic of those associated with psorosis.

In 1941 Klotz and Fawcett (3) noted that infectious variegation had been found a few times associated with the crinkly leaf condition of lemons. They transmitted both disorders by budding to healthy lemon, sweet orange, and sour orange trees. In their descriptions of psorosis "A", psorosis "B", concave gum, blind pockets, crinkly leaf, and infectious variegation Fawcett and Bitancourt (1) in 1943 referred to the first five as "varieties" of psorosis. They were uncertain whether infectious variegation differed sufficiently from crinkly leaf to permit its being described as a distinct "variety."

In a review of information on infectious variegation Wallace and Grant (6) in 1953 noted that the white or pale yellow areas occurred and persisted in the leaves of grapefruit as well as in those of lemon and sour orange. In 1957 Wallace (5) reported the results of cross-protection tests in which "sweet orange seedlings were inoculated and infected singly with concave gum, blind pocket, crinkly leaf, and infectious variegation. When such seedlings were reinoculated by means of lesion bark, they displayed the same protection phenomenon described for psorosis 'A'. Thus relationship of the various psorosis virus strains was demonstrated experimentally."

The purpose of the present paper is to report the finding of infectious variegation in Florida and the results of tissue transmission tests.

After the severe, cold weather in Florida in 1957 all the citrus experimental plantings of the United States Department of Agriculture Horticultural Station were examined to determine the degree of damage in each tree. During recurrent visits to the Cherry Lake fertilizer plots, two Marsh grapefruit trees with abnormal chlorotic foliage on a few individual branches were noted[2]. A photograph of a representative branch and fruit with and without symptoms is shown in Figure 1.

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[2] The first collection of the abnormal branch material was made by L. C. Cochran, Paul Smith, and Walter Reuther. Subsequent collections were made by the authors.

FIGURE 1. Marsh grapefruit branch and fruit samples from a single field tree. Left fruit shows irregular yellow-green blotch spots. Lower left branch shows yellow leaves with green spots or green leaf with irregular yellow areas. Center branch shows extreme pocketing, bumps and malformed leaves. Right fruit and branch are from an apparently healthy branch collected from the same tree.

FIGURE 2. The two leaves at left are Duncan grapefruit, one healthy and one showing the irregular white blotches that occurred following inoculation and growth in a nursery. The two leaves at right are from infected Eureka lemon plants that showed the vein and veinlet banding and blotch symptoms under greenhouse conditions.

Tissue transmission tests were undertaken with buds or leaf pieces from the branch or foliage showing symptoms. Three to five potted seedling plants of each of several different citrus varieties were inoculated and a comparable number of non-inoculated control plants were maintained under greenhouse conditions for 4 months and then transplanted to a nursery. The results are presented in Table 1, and the symptoms produced on leaves of inoculated plants are shown in Figure 2.

Procedures to stimulate variegation-symptom expression were undertaken following observations made on the first series of plants inoculated in the greenhouse. Sometimes all three or five plants in a series did not show the same degree of symptoms and in some cases the symptoms occurred on one branch and not on others. Also, most plants tended to put out new growth that showed few or no symptoms.

Twenty-four Eureka lemon plants 18 to 20 inches in height were selected for apparent uniformity. These were divided into four groups of six plants each. Plants in group 1 were cut back to a 10-inch height and all except three leaves at the top of the cut main stem were removed. Plants 1 to 4 were inoculated by two leaf-piece grafts from a standard source of variegation; 5 and 6 served as control. Plants in group 2 were treated in a similar manner except that all leaves were left on the main stem. Plants in group 3 were treated in a similar manner except that all leaves were removed from the top 4 inches of the main stem. This left several leaves on the lower 6 inches of main stem below the points of inoculation. Plants in group 4 were not cut back and only one or two leaves were removed to facilitate insertion of the leaf-piece inoculum.

In all groups only a single bud immediately above the leaf-piece inoculum was allowed to develop. All other new shoots were removed shortly after breaking bud.

The new growth on the four inoculated plants in group 3 all showed distinct variegation symptoms in 14 days. The inoculated plants in group 1 showed symptoms on the new growth in 17 days. In group 2 there were three plants that showed symptoms in 17 days, but one plant did not show symptoms for a month. None of the plants in group 4 showed symptoms for over 2 months, at which time defoliation and cutting back were carried out to try to stimulate symptoms. All four inoculated plants in group 4 finally showed symptoms following the shock treatments of defoliation and pruning.

Table 1. Symptoms on different citrus varieties following inoculation by bud or leaf tissue graft from the Marsh grapefruit branch or foliage with infectious variegation.

Citrus variety	Leaf symptoms on plants held in: greenhouse 4 months	nursery 9 months[a]
Duncan grapefruit	vein banding, crinkle	irregular white blotches, stunting
Sour orange	vein banding, yellow spots, crinkle	slight crinkle, stunting
Eureka lemon	vein banding, speckle, blotch, crinkle	yellow blotch, crinkle, stunting
Ponderosa lemon	vein banding, severe crinkle	crinkle, stunting, yellowing
Rangpur lime	vein banding, oak-leaf pattern, twist, crinkle	crinkle, stunting, yellowing
Citrus sp. Moi	vein banding, yellow spot, crinkle	white-yellow spots, crinkle
Hamlin orange	veinlet banding, psorosis slight blotch, crinkle	Hamlin no symptoms, but branch of grapefruit from bud inoculation showed white blotches
Calamondin	Typical psorosis symptoms	None

[a] Control plants for all varieties remained free of symptoms.

As a result of this experience, a standardized procedure was adopted, the plants to be inoculated were cut back to leave a 6- to 8-inch main stem with one or two leaves at the top. The inoculum was placed in the stem immediately below the attached leaf. As soon as sprout growth of the host started, in 6 to 10 days, the one or two old leaves and all sprouts except the one immediately above the inoculum were removed. When this procedure is followed, some variegation symptoms have been expressed on such citrus species as Columbia sweet lime, Orlando tangelo, Key lime, and Alamoen, as well as on grapefruit, sour orange, and Eureka lemon seedlings.

DISCUSSION

On the basis of field tree symptoms and those obtained by tissue inoculation of citrus seedlings in the greenhouse, the infectious variegation found in Florida appears to be similar to that described in California and Sicily. The variegation as tested in Florida produced crinkle leaf and veinlet-banding symptoms as well as irregular blotched areas. Thus it may be judged that the field trees were infected with two or more strains of psorosis virus.

The two infected grapefruit trees that showed variegation on some branches at Cherry Lake are now 18 years old. The new foliage developed in the past 2 years has a generally healthy appearance. Only one leaf with white blotched areas and a number of crinkled and warped leaves were found during a recent inspection of the trees. Scaly-bark symptoms are not yet common in this orchard, but two trees of 700 have developed bark symptoms in the past 2 years. The trees which show these localized psorosis bark symptoms are not the same two trees that showed variegation. Crinkled leaves and varying degrees of stunting suggest that possibly additional trees in the plots are also carriers of the variegation-psorosis virus complex, but no distinctive white-blotched areas were found in the leaves.

Defoliation of the grapefruit trees, following the cold weather in 1957, would appear to have been an important factor in stimulating symptom expression which led to the initial discovery of the two infected field trees. Under greenhouse conditions the shock treatments of defoliation and cutting back of inoculated plants stimulated symptom expression.

Under field and greenhouse conditions the infected plants tend to grow out of variegated symptom expression. This suggests that the psorosis virus complex responsible for variegation may be present in many psorosis virus-infected field trees but that variegation symptoms are seldom seen except under conditions of stress or shock and even then these symptoms may be limited to a few branches or leaves on a given tree.

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OCCURRENCE OF THE SCLEROTIAL STATE OF CIBORINIA CANDOLLEANA (LÉV.) WHET. IN THE UNITED STATES OF AMERICA

Lekh R. Batra[1] and J. M. Staley[2]

Ciborinia candolleana (Lév.) Whetzel (= Peziza candolleana Lév. = Sclerotinia candolleana (Lév.) Fuckel = Sclerotium pustula Fr.) has been reported attacking several species of oak in England, Scotland, Denmark, Germany, France, and Switzerland. Wilson and Waldie (3) reported an oak disease causing death of leaves and, in some cases, premature defoliation, from several localities in Scotland over the period of 1925-1927. These workers made observations on Quercus pedunculata, Q. sessiliflora, and Q. rubra, and attributed the cause of their foliar disease to Sclerotinia candolleana. They also mentioned Q. robur and Castanea sativa as being susceptible.

Despite many attempts over a period of 30 years, Whetzel was unable to find this fungus in the United States of America. He had several collections of the organism from Scotland, Denmark, and other European countries. Rehm's herbarium at Stockholm contains two small apothecia and one sclerotium of the Sclerotium pustula-type which were collected by B. O. Dodge from Madison, Wisconsin, in 1909 (Whetzel's notes at Cornell University). No further information is available to the writers as to the identity of this collection.

During the summer of 1959 Ciborinia candolleana was isolated in its sclerotial form from the veins of red oak, Quercus rubra, that had been partially damaged by an oak leaf roller, Argyrotoxa semipurpurana Kearfott[3]. It was also isolated from the blackened region of partially dead twigs. Both types of material were collected from the Tiadaghton State Forest near Slate Run, Pennsylvania.

The fungus is characterized in culture by small, black, shiny sclerotia with a scanty aerial mycelium. The loaf-shaped sclerotia are formed with the agar medium and become erumpent

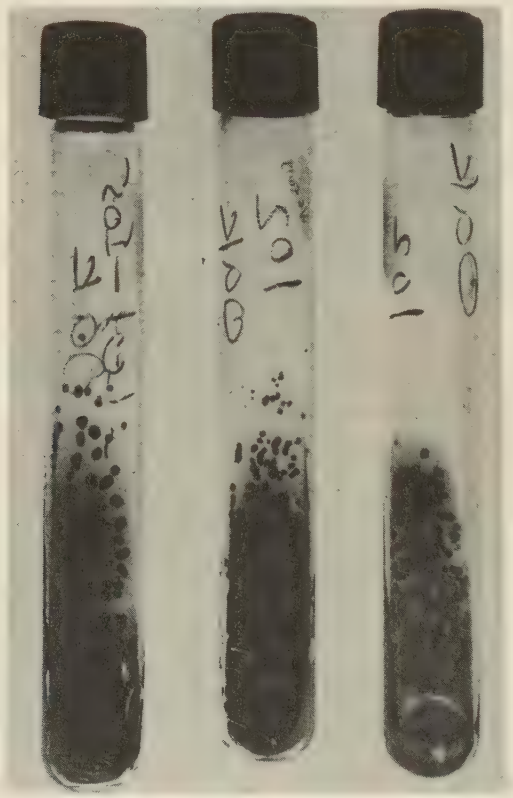

FIGURE 1. Sclerotia formed in agar culture of Ciborinia candolleana, somewhat reduced.

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[2]Pathologist, U. S. Forest Service, Northeastern Forest Experiment Station.

[3]The genus Argyrotoxa is treated as Croesia Hubner in recent European literature. The authors thank Dr. J. G. Franclemont for his identification and proper citations.

FIGURE 2. Sclerotia formed in agar culture of an unidentified member of the Sclerotiniaceae isolated from leaves of Quercus prinus, somewhat reduced.

subsequently (Fig. 1). Spermatia are formed in culture, but all attempts to produce apothecia from sclerotia by methods reported elsewhere (1, 2) have so far been unsuccessful. It is suspected that a period of dormancy may be one of the factors in apothecial formation. Further studies are being conducted at Swarthmore College.

Another member of the Sclerotiniaceae was also collected in the apothecial state from a previous year's leaves of the chestnut oak, Quercus prinus, in early August from Logan State Forest near State College, Pennsylvania. Ascospore shootings gave cultures with a cottony mycelium and thin, substratal types of sclerotia (Fig. 2). The identity of this second fungus is unknown at present. It is possible that this fungus may also be referred to the genus Ciborinia Whetzel or the genus Rutstroemia Karsten emend. Rehm in the future. No apothecia have been produced from the sclerotia obtained in culture as yet.

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RESISTANCE TO BACTERIAL WILT IN EGGPLANT IN NORTH CAROLINA

N. N. Winstead and Arthur Kelman[1]

Summary

In both greenhouse and field studies the Matale and Kopek eggplant varieties have good resistance to Pseudomonas solanacearum. No plants of these varieties became diseased in a field test; in contrast all Black Beauty and New York Spineless plants were killed. Florida Market, Florida High Bush, and Fort Myers Market eggplants were intermediate in resistance. The undesirable shape and color of the fruits of Matale and Kopek will probably limit their acceptance for commercial eggplant production in North Carolina, but their superior resistance may be of great importance in a breeding program.

Bacterial wilt, caused by Pseudomonas solanacearum E. F. Sm., is an important disease of eggplant (Solanum melongena) in many parts of the world (5). In North Carolina field losses of 50% have been observed by mid-summer. Initial evaluations of eggplant varieties for resistance to bacterial wilt were made in Puerto Rico (7). The varieties Black Beauty, Excelsior and New York Spineless were susceptible to bacterial wilt; whereas, native varieties Long Green and Camuy were resistant. The Puerto Rican varieties Long Green and Camuy were also resistant to bacterial wilt in Ceylon (3); however, the use of these varieties in these two areas was limited because they lacked desirable qualities. A breeding program was initiated in Puerto Rico to develop a resistant eggplant variety with acceptable quality (2, 12). From this program new high yielding varieties Puerto Rican Beauty, Rosita and E-12 were developed that performed well in Puerto Rico (13, 14, 15). Subsequently, two of these varieties, Puerto Rican Beauty and E-12, were tested in South Africa and both were susceptible under severe disease conditions (17, 18). In the same tests plants of the Matale variety obtained from Ceylon were moderately resistant, and two Javanese varieties, Terong Kopek and Terong Gowok, were highly resistant. Crosses were made between the Matale and the Javanese varieties. Resistant varieties obtained from these crosses were released by the Botanic Station (4). Reports of resistant varieties of eggplants have also come from the Philippines (9) and Malaya (1). Workers in Ceylon originally had described the Matale variety of eggplant, which they found to be practically immune to P. solanacearum in that area. The seed source for this variety was in Matale South, where this variety had been grown for a number of years (10, 11). Kunieda (6) reported the development of varieties with resistance to bacterial wilt in Japan and investigated the mechanism of disease resistance in these resistant varieties. According to reports from Japan a number of wilt resistant varieties are presently available there.

Because of the seriousness of the disease on eggplant in North Carolina, studies were initiated to determine whether the strain of the wilt bacterium in this area would affect varieties that have shown superior resistance in other parts of the world.

MATERIALS AND METHODS

Two varieties reported as resistant to P. solanacearum in South Africa were obtained from Dr. Vincent A. Wager of the Botanical Station at Durban, Union of South Africa for use in these tests. In 1942 Dr. Wager had obtained the variety Matale from Peradenia, Ceylon and the variety Kopek from Buitenzorg (Bogor), Java. These two varieties were compared with commercial varieties in greenhouse and field tests to determine their relative resistance to P. solanacearum under field and greenhouse conditions in North Carolina.

Plants were grown in 6-inch pots or 14 x 21 x 4 inch metal flats for greenhouse tests and inoculated by the root cutting or stem puncture techniques (19). In most tests plants had 8 to 10 expanded leaves at the time of inoculation. Varieties tested in the greenhouse were Black Beauty, Florida Market, Matale and Kopek. In addition to these four varieties, Florida High Bush, Fort Myers Market, and New York Spineless were tested in the field. Field evaluations at the North Carolina Experiment Station at Faison, North Carolina were made in a plot where Black Beauty eggplant had been planted continuously for the preceding 6 years, and 95 to 100%

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of the plants of this susceptible variety died in the sixth year.

EXPERIMENTAL RESULTS

Disease severity was usually greater in greenhouse than in field tests. All Black Beauty plants were killed 10 days after inoculation in greenhouse inoculation tests irrespective of inoculation techniques. Florida Market plants also died but wilting developed 2 to 4 days later than in Black Beauty and the plants died 14 to 21 days after inoculation. Matale and Kopek plants rarely succumbed except when small succulent plants (3 to 4 leaves) were inoculated. In one test, in which small plants were inoculated by the root cutting technique, 45% of the plants of each of these varieties were dead within 10 days after inoculation; however, in this same test 100% of the Black Beauty and Florida Market plants were killed.

The four varieties differed in extent of vascular browning following inoculation. In larger root-inoculated plants, vascular browning was observed in all Black Beauty and most Florida Market plants 7 days after inoculation. The extent of vascular browning was much less, however, in Florida Market than in Black Beauty plants. Spread of vascular browning from the point of inoculation in Black Beauty plants was more rapid than that in Florida Market; however, vascular browning was usually extensive in plants of both varieties. Some vascular browning was observed in all Matale and Kopek plants inoculated by the stem puncture technique; however, the vascular browning was always limited in extent, except when very small young plants were inoculated.

Since Matale and Kopek appeared to be resistant, Black Beauty susceptible, and Florida Market slightly less susceptible than Black Beauty in greenhouse inoculation tests, these and three additional varieties, Florida High Bush, Fort Myers Market, and New York Spineless, were compared for resistance in field tests. Eighty plants of each variety were planted in the field in a randomized block with four replicates early in May. Observations were made at 2-week intervals during the growing season to determine whether bacterial wilt was the cause of death. The first plants showing wilt symptoms were observed on July 2. By the end of the season (mid-August) the following percentages of plants were affected including dead and wilted plants: Matale, 0; Kopek, 0; Florida Market, 51; Fort Myers Market, 75; Florida High Bush, 78; New York Spineless, 100; and Black Beauty, 100. When these data were analyzed statistically the varieties fell into four resistance classes based on L.S.D. 0.01 values. These classes were: 1) Matale and Kopek, resistant; 2) Florida Market, moderately resistant; 3) Fort Myers Market and Florida High Bush, moderately susceptible; and 4) New York Spineless and Black Beauty, susceptible.

DISCUSSION

Although Florida Market, the eggplant variety most widely grown in North Carolina, appears to carry a moderate level of tolerance to bacterial wilt in comparison with varieties such as Black Beauty, 50% less in commercial fields is not uncommon. This report also confirms a previous report (13) that Florida High Bush was more resistant than Black Beauty. The resistance in Matale and Kopek appears adequate for commercial eggplant production in P. solanacearum infested soils; however, neither variety has fruit that is acceptable at present to the North Carolina markets. The objectionable features are considered to be associated with appearance rather than with table quality. In North Carolina field tests the fruits are essentially similar to the description given by Wager (18). Matale fruits are 6 inches long and 3 to 4 inches thick, and dark purple in color. Matale fruits appear light purple when compared with the almost black fruits of Florida Market. Kopek fruits are bluish-purple and lighter than Matale fruits in color, cylindrical, 6 to 10 inches long, and 3 inches thick.

The observations on the high resistance of Matale and Kopek under North Carolina conditions, as well as in many other areas of the world, is of significance in terms of the similarity of reaction of these varieties to bacterial wilt under diverse environmental conditions. The Matale variety is resistant to P. solanacearum in Ceylon (11), the Union of South Africa (18), and in North Carolina. Similarly, the Kopek variety is resistant in Java, Union of South Africa (18), and North Carolina. Thus, in these widely separated geographic areas, eggplant varieties have shown similar high levels of resistance to the strains of P. solanacearum indigenous to the areas. Although there is evidence of wide variation in the ability of strains of P. solanacearum to attack certain solanaceous hosts (5), these data support the conclusion that varieties developed for resistance in one area may have utility in other parts of the world. This relationship has held for the wilt-resistant Schwarz 21 peanut in Indonesia (16) and in North

Carolina (19), and for wilt-resistant tobacco varieties grown in North Carolina and Japan (5, 8).

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OCCURRENCE OF VERTICILLIUM WILT ON PEANUTS[1]

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In 1958 a vascular disease was collected on peanuts in Roosevelt County, New Mexico, from a field where Verticillium wilt on cotton occurred in 1957. Isolations gave the microsclerotial type of Verticillium albo-atrum Reinke & Berth. In 1959 Verticillium diseased peanuts were collected in this area on five fields, occurring mainly on single plants. However, at the lower end of one field, where high soil moisture resulted from over-irrigation, 25% of the plants were killed or badly stunted by Verticillium. In Roosevelt County, Verticillium wilt occurs generally on cotton but causes little loss to this crop.

Symptoms on peanuts began to develop at the start of flowering on naturally infected plants in the field. Localized yellowing and withering of leaflets were the first symptoms. After these foliage effects became general, defoliation occurred and brown discoloration of the stele was observed at or below the soil line. These symptoms are easily mistaken for combined effects of stem rot and leaf spot diseases. However, the characteristic symptom is discoloration of the stele in the absence of surface lesions on the root crown and stems.

Verticillium was isolated by plating sections of discolored stele on potato-dextrose-carrot agar fortified with streptomycin sulfate or nitrate. Inoculations were made by dipping the roots of 7 to 10 day old seedlings in macerated cultures grown on autoclaved oats. Isolates of Verticillium from cotton, peanuts, chili pepper and Solanum elaeagnifolium were inoculated onto cotton (1517C) and peanuts (N. M. Valencia), using three seedlings of each host to test each isolate. After 3 weeks leaf yellowing or kill of plants had developed on cotton and peanuts from inoculations with all cultures. It appears that, on inoculation, peanuts are susceptible to Verticillium from a wide range of plant material.

Verticillium on peanuts was reported in combination with Fusarium sp. from Asia[3] and Australia[4]. The results reported here show that V. albo-atrum alone can parasitize peanuts.

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DIFFERENTIAL TRANSMISSION OF FOUR STRAINS OF STRAWBERRY VEIN BANDING VIRUS BY FOUR APHID VECTORS

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Abstract

Four strains of vein banding virus were transmitted by each of the four aphid vectors Pentatrichopus thomasi, P. jacobi, P. fragaefolii, and an atypical clone of P. fragaefolii with the one exception that the atypical clone failed to transmit the type strain of the virus. A previous report that P. thomasi (sensu stricto) is not a vector of vein banding virus is therefore in error, the non-vector being the atypical clone of P. fragaefolii.

Non-transmission of strawberry vein banding virus by two of four aphid colonies of Pentatrichopus fragaefolii (Ckll.) was reported by Frazier (1). Later the two non-transmitting colonies were cited by Frazier and Posnette (2) as having been determined to represent the species P. thomasi Hille Ris Lambers, but this report must be modified in the light of recent studies by Schaefers (3).

It is the purpose of the present paper to clarify the previous reports with evidence resulting from transmission tests using four strains of vein banding virus and four Pentatrichopus aphid vectors.

MATERIAL AND METHODS

Aphid Vectors: Using the number of setae (range 0-8) present in the second marginal rows on the anterior abdominal tergites as the most divergent character and examining large numbers of aphids, Schaefers (3) found three Pentatrichopus species in California with differing setal characteristics (range, mean and mode, respectively) as follows: P. thomasi; 0-8, 7 and 8. P. jacobi (Hille Ris Lambers); 0-8, 3.8 and 5. P. fragaefolii; 0-1, .04 and 0, agreeing closely with specimens examined from New York and Europe. In addition, he distinguished a population of aphids found in a greenhouse in Berkeley having a range of 0-6, a mean of 1.1 and a mode of 0 setae. Because, like P. fragaefolii, it possessed a modal class of zero setae he referred it to that species although he pointed out that its setal range of 0-6 differed markedly from the 0-1 range of P. fragaefolii. In the latter respect the greenhouse population approaches the 0-8 setal ranges of both P. thomasi and P. jacobi but, also lacking the darkened terga characteristic of P. jacobi viviparae, the population appears to be atypical of any of the three species and might represent a variety of any one or an entity equally distinct. For present purposes, and lacking other identification, the aphids representative of the Berkeley greenhouse population will be referred to as the atypical clone of P. fragaefolii.

Colonies of each of the above four Pentatrichopus entities were kindly supplied to the present author by Dr. Schaefers and were used for the tests reported herein.

Viruses: It is assumed that the four viruses used in these tests represent different strains of the strawberry vein banding virus, namely, yellow vein banding virus (Frazier and Posnette (2)), vein banding virus (Frazier (1)), and two new strains, chiloensis and eastern vein banding virus. The four strains induce the same general type of symptoms which, however, differ considerably in the severity of expression in the order of the strains listed above from the most to the least severe.

In conducting the tests, access feeding periods of about 17 hours on the inoculum plants and 22 hours on the test plants were used. In any one test the four virus strains were compared simultaneously for transmission by a single vector using 10 aphids per test plant and five test plants for each virus. The tests were replicated six times for each vector. No simultaneous comparison of more than one vector was made so the results do not reflect relative vector efficiency. The tests were carried out during all seasons of the year and many indicator plant varieties of Fragaria vesca were used.

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RESULTS

Results of the tests are given in Table 1. It is evident that P. thomasi is a vector of all four strains of vein banding virus, as are also P. jacobi and P. fragaefolii. It is equally evident that the atypical clone of P. fragaefolii failed to transmit the type virus but did transmit each of the other three strains.

Table 1. Results[a] from tests of transmission of four strains of strawberry vein banding virus by four Pentatrichopus aphid vectors to Fragaria vesca test plants using 10 aphids per plant and acquisition and test access feeding periods of about 17 and 22 hours respectively.

Aphid vector	Virus strain			
	type	yellow	chiloensis	eastern
fragaefolii (atypical)	0/30	26/30	20/30	26/30
fragaefolii	27/30	15/30	25/30	29/30
thomasi	19/30	19/30	29/30	23/30
jacobi	15/15	12/15	11/15	8/15

[a]The numerator denotes the number of plants infected; the denominator the total number of plants fed upon by test aphids in replicates of five plants.

Examination of a few preserved slide specimens of the previously reported non-transmitting colonies (Frazier (1)) indicated that they could be identified as either the atypical clone of P. fragaefolii or as P. thomasi, since there is a considerable overlapping in the setal ranges of the two aphid entities (0-6 and 0-8, respectively). Since the colonies also were similar to the atypical clone of P. fragaefolii in the fact that they originated in the Berkeley area and neither transmitted vein banding virus, it seems most probable that they represented populations of the atypical clone of P. fragaefolii rather than P. thomasi sensu stricto with its demonstrated ability to transmit the virus.

Taxonomically, the four aphid vectors might equally represent four species, merely a single species, or some intermediate degree of relationship. The four viruses do not appear to represent more than strains of a single species, and if such is the case then the fact that one of the aphid entities apparently cannot normally transmit one of the virus entities may indicate that differences in vector-virus combinations at a sub-specific level can be great enough to predicate a vector or non-vector relationship.

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BEHAVIOR OF SOME BARTLETT PEAR TREES ON THEIR OWN ROOTS

Earle C. Blodgett and Murit D. Aichele[1]

A natural result of research on pear decline since 1955 has been the increased interest in the pear rootstock situation in Washington (1, 2, 3, 4, 6). The writers have made special efforts to study this problem and to learn more about the roots under both healthy and sick pear trees.

In the October 23, 1958 issue of The Goodfruit Grower there appeared an article entitled "Mystery shrouds one-acre pear planting near Zillah." The orchard described belongs to Lars Henriksen who purchased the ranch from the late Ed Chenour. This orchard was said to have been planted more than 50 years ago on quince roots with the 300 trees planted 12 feet on the square. So far as the writers are aware this is the only old Bartlett orchard left in central Washington which is said to have been planted on quince roots. Blight has not been severe at any time and production has been far above average: in 1958, 26 tons; in 1959, 25 tons; and several years ago 50 tons were said to have been harvested from the 1 acre.

We examined the orchard in November 1958 and with moderate digging around the base of several trees could find no external evidence of a stock-scion union. While tree growth was not abundant, the foliage was generally good. Tree growth ratings of 1 to 5, as used in the pear decline survey, were made in 1959. A normal, vigorous tree is rated 1 and trees having no growth rated 5. These data are shown in Table 1 under orchard 1 A, B, C. There has been no evidence of quick decline in this orchard.

On May 4, 1959 we made a more detailed survey of the block and found suckers from a rootstock under only two trees, both Winter Nelis pollinizers. Under many of the trees shoots were arising from the crown and main roots which were all identified as Bartlett. Careful excavation was made under four of the original trees. One of these, a dead stump, probably Winter Nelis, appeared to have been grafted on a pear seedling. In the other three trees, all bearing, there was unmistakable evidence that the Bartlett trees had been propagated originally on quince root and subsequently have scion rooted. The old quince portion was dead but still intact under the crown of the trees, as shown in Figure 1. A similar situation is pictured by Day (5) in California. More detailed evidence of the original graft is shown in Figure 2.

FIGURE 1. In the center is the original quince root graft which is dead. On both sides are roots arising from the Bartlett top.

FIGURE 2. Details of the quince Bartlett graft union shown in Figure 1.

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Table 1. Growth ratings of pear trees in four orchards in the Zillah and Selah areas of Washington, 1959.

Orchard	Total trees	Age (approx.)	Growth rating 1	2	3	4	5	Remarks
1 A, Bartlett	253	50+	0	13	201	37	2	All these trees were said to be originally on quince. They are now believed to be entirely own rooted.
1 B, Winter Nelis pollinizers	24	50+	2	6	15	1	0	These are believed to be on seedling roots.
1 C, Bartlett replants	30	2-12	8	3	15	4	0	These are believed to be on Bartlett seedling roots.
2 A, Bartlett	98	9	87	7	4	0	0	These were said to be originally on quince. Most are now believed to be partly or completely own rooted.
2 B, Bartlett replant	6	2-8	4	2	0	0	0	These are believed to be on Bartlett seedling roots.
3, Bartlett	29	30	0	1	18	6	4	Oriental rootstock. Some of the trees are known to have self rooted. These 29 trees are along the lower edge of the orchard and are the only remaining trees in a 4-acre planting. The orchard was removed primarily because of decline.
4 A, Bartlett	3	25	3	0	0	0	0	These are believed to be on French root and are in a 3-acre orchard of similar trees.
4 B, Bartlett	3	25	0	0	0	3	0	These are believed to be originally on Oriental root but are known to be partially own rooted. They are part of a 3-acre orchard of similar trees.

Since there is a hardpan layer about 15 inches below the soil surface, the original planting was probably made as deep as possible in order to anchor the trees. It is common knowledge that Bartlett propagated directly on quince ordinarily does not produce a good union and scion rooting occurs if there is sufficient opportunity. There is little doubt that the trees soon scion rooted and choked out the quince. In this orchard one quince tree grew for several years in a pear tree position. Evidently that was the only quince root to survive in the entire planting.

All evidence indicates that this 1-acre orchard which has borne so prolifically over the years is on Bartlett root and that there is now no stock union. We believe the evidence presented adequately explains this interesting "mystery."

In 1957, while the stock scion unions in various Bartlett pear orchards in the Yakima Valley were being examined, several trees were found to have scion rooted. This probably took place in one case (orchard 3, Table 1) because soil built up around the tree crowns in the lower part of the orchard. In another case (orchard 4 B) the trees apparently were planted extra deep. In general these own-rooted trees have maintained better tree growth than most others in the same orchard. In orchard 4 B, however, the three own-rooted trees are in very poor condition. They represent several rows of trees on oriental root, adjacent to a similar age planting (orchard 4 A) on French root, as evidenced by typical suckers.

In reply to an inquiry through The Goodfruit Grower, one orchardist at Zillah said that he had a 9-year-old planting of Bartlett on quince. The orchard was examined carefully and tree ratings were made as shown in Table 1, orchard 2 A and 2 B. Most of the trees are in the process of scion rooting, judging by observations on quince suckers and limited examination of Bartlett roots at the crowns.

These records and observations on Bartlett trees on their own roots open up new, valuable leads and opportunities in pear decline studies which are being followed.

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REACTION OF SIXTEEN VARIETIES OF ALFALFA TO TWO SPECIES OF ROOT-KNOT NEMATODES[1]

Harold W. Reynolds and John H. O'Bannon[2]

Summary

Measured inoculum of two species of root-knot nematodes, Meloidogyne javanica javanica and M. incognita acrita, was tested on 16 varieties of alfalfa in replicated tests in the greenhouse. All varieties were infected by each of the two species of root-knot nematodes. Three varieties (African, Moapa and India) were highly resistant to both species of root-knot nematodes. In addition, the variety Moapa was resistant to the spotted alfalfa aphid. The Javanese root-knot nematode was more damaging to most of the varieties than was the cotton root-knot nematode.

Two species of root-knot nematode, Javanese (Meloidogyne javanica javanica (Treub) Chitwood) and cotton (M. incognita acrita Chitwood), are commonly found infecting alfalfa in Arizona. The degrees of infection and damage to the plants vary from light to heavy depending upon the variety of alfalfa grown, soil texture, and cropping practices. Apparently soil texture is of considerable importance, as most severe infection occurs in sandy soils. Cropping practices or crops grown prior to seeding alfalfa are also of importance; if root-knot-susceptible crops are grown the initial inoculum and rate of root-knot buildup will be faster than if less susceptible crops are grown. If root-knot-susceptible alfalfa varieties are grown and high root-knot nematode populations occur, susceptible crops such as cotton and melons following in the rotation will sustain losses. Generally the northern (hardy) varieties or selections of alfalfa are more susceptible to these two species of root-knot nematodes than the southern (non hardy) ones (4).

Infection of the primary roots by root-knot nematodes often occurs in the seedling stage. As the primary root is damaged lateral roots arise near the point of infection; these in turn are attacked, which results in a marked reduction in length and number of roots and thereby a reduction in the ability of the plants to forage. Thorne (7) observed that in root-knot nematode infested fields alfalfa stands did not become well established. The root systems were branched and spreading, and few long tap roots were developed. The nematode does not always cause conspicuous galling on alfalfa roots as it does on certain other hosts. Therefore, a close examination is generally necessary to reveal the presence of the pest. Willhite and Smith (8) observed that heavy root-knot infections influenced the type of root system and the quantity and quality of forage produced.

Previous work (4, 6) has been done relative to the range of susceptibility of several northern and southern varieties of alfalfa to root-knot nematodes. Because of increased interest in the role of insects in alfalfa forage and seed production, some promising varieties are under observation for insect resistance. Selections of these varieties[3] from the standpoint of insect resistance were studied to determine varietal reaction in the presence of two species of root-knot nematodes.

MATERIALS AND METHODS

One hundred twenty-eight 4-inch pots containing soil free of root-knot nematodes were embedded in sand in the greenhouse bench. To provide uniform heat and optimum temperature for development of the root-knot nematode, a mean temperature of 27° C was maintained by means of an electric heating cable embedded in sand beneath the pots. Sixty-four pots were inoculated with the Javanese root-knot nematode and 64 with the cotton root-knot nematode. The inoculum which consisted of viable larvae in water was prepared in accordance with the procedure outlined by Godfrey (1). Approximately 1800 larvae were added to the soil of each pot just before the alfalfa was seeded. Each group of 64 pots were spaced sufficiently far from the other group to prevent contamination through watering or larval migration.

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[1]Cooperative investigations of the Crops Research Division, Agricultural Research Service, United States Department of Agriculture and the Arizona Agricultural Experiment Station.

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[3]Acknowledgment is given to M. W. Nielson, Entomologist, Entomology Research Division, Agricultural Research Service, Mesa, Arziona, who supplied seed of the varieties studied and observations on reactions to insects.

Each group of 64 pots were seeded with 16 varieties of alfalfa and randomized in four blocks. Approximately 12 plants were grown in each pot for 45 days. The plants were harvested and washed lightly to remove excess soil. Each plant was given a root-knot rating as described by Smith and Taylor (5). Root-knot indices were calculated by the McKinney (3) formula as modified by Horsfall and Heuberger (2), who applied it to a defoliation disease of tomatoes. Rating for each plant was determined with aid of the dissecting microscope.

RESULTS AND DISCUSSION

As shown in Table 1, all varieties of alfalfa tested were infected by each species of root-knot nematode, with infection varying from slight to moderate.

Table 1. Susceptibility of 16 varieties of alfalfa to two species of root-knot nematodes.

Variety	Root-knot index[a] (mean of four replications)	
	Javanese root knot	Cotton root knot
African	3.0 a[b]	3.0 a
Moapa	7.0 a	2.8 a
India	6.3 a	4.0 a
Hairy Peruvian	34.8 cde	6.5 a
Sirsa #9	20.5 b	7.0 ab
North Carolina Synthetic A(51)5	39.5 def	11.8 abc
Chilean 21-5	43.3 ef	17.0 bcd
Du Puits	32.8 bcde	18.3 cd
Caliverde	46.3 efg	22.3 cde
Argentine	48.8 fgh	23.8 de
Zia	60.8 hi	24.5 de
Hardigan	29.3 bcd	24.8 de
Lahontan	62.8 i	27.0 def
Doehnfeldt	25.8 bc	30.3 efg
Ranger	64.3 i	37.0 fg
Nevada Synthetic F	57.3 ghi	40.5 g

[a] Root-knot index: 0 = no infection; 1-25 = very light infection; 26-50 = light infection; 51-75 = moderate infection; 76-100 = heavy infection.

[b] Values followed by the same letter are not significantly different according to Duncan's Multiple Range Test at the 5% level.

The degree of infection of essentially all varieties was greater by the Javanese root-knot nematode than by the cotton root-knot nematode. Three varieties (African, Moapa and India) were highly resistant to both species. These studies indicate that at least three varieties of alfalfa can be used in crop rotations where either of these two root-knot nematode species is present. The variety Moapa, however, has the advantage of being resistant to the spotted alfalfa aphid, Therioaphis maculata (Buckton), a troublesome alfalfa pest in the southwest.

The importance of alfalfa from an economic standpoint and as a soil-fertility builder cannot be overemphasized. Consequently, the use of varieties which possess resistance to certain destructive pests aids in development of a successful cropping program.

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CROPS RESEARCH DIVISION, AGRICULTURAL RESEARCH SERVICE, UNITED STATES DEPARTMENT OF AGRICULTURE AND THE ARIZONA AGRICULTURAL EXPERIMENT STATION

EFFECTIVENESS OF CERTAIN FUNGICIDES AND GIBBERELLIC ACID AS SEED TREATMENT FOR PEARL MILLET

Akhtar Husain[1]

Abstract

Ten different seeddressing fungicides were tested for their effectiveness in improving germination of pearl millet seeds. With the exception of Leytosol, which was toxic, all the chemicals increased the germination percentage to a considerable extent. Ceresan wet, Spergon, captan, Dithane Z-78, Panogen-15 and Arasan were found to be more effective than others. Addition of gibberellic acid to captan and Ceresan did not result in any significant increase in germination, but the height of the seedling was increased.

Poor germination and thin crop stand are some of the common causes of crop failure in India. Pearl millet suffers from this trouble to a certain extent in almost all areas where this crop is grown. In a majority of cases the trouble arises from infection by seed- or soil-borne fungi which cause seedling disease. As seed treatment has not become a very popular feature of crop production in this country, no recommended seeddressing fungicides are available which have been found effective for this crop.

In recent years it has been suggested that gibberellic acid may enhance the value of the seed protectants, if it is used in conjunction with fungicides for treatment of the seeds of various crops. It has been argued that accelerated emergence of seedlings caused by gibberellic acid treatment will reduce the period when the plants are most vulnerable to the attack of soil fungi[2, 3].

A number of seeddressing fungicides were tested for suitability in treating pearl millet seeds. Gibberellic acid was also included to test its synergistic effect as seed protectant for this crop.

The paper presents a preliminary report of the results obtained in these experiments.

MATERIALS AND METHODS

Ten different fungicides and a local variety of pearl millet were used in these experiments which were carried out during the months of August and September.

The different fungicides employed in the test were: Ceresan (ethyl mercury chloride), Leytosol (phenyl mercury urea), Leytosan (phenyl mercury urea), Spergon (tetrachloro-p-benzoquinone), captan (Flit-406), (N-trichloromethylthio-4-cyclohexene-1, 2 dicarboxymide), Dithane Z-78 (Zinc ethylene bisdithiocarbamate), Panogen-15 (phenyl mercury dicyandiamide), Arasan (tetramethyl thiuram disulphide), and Phygon (2, 3-dichloro-1, 4-naphthoquinone).

The seeds were treated in small lots in a round bottle using the amount of chemical recommended by the manufacturers. To test the effect of gibberellic acid, a special dust formulation of the hormone (Gibrel-88, Merck. & Co.) was mixed with Ceresan and captan at the rate of 8 ounces per 100 pounds of seed.

Fifty seeds of each treatment were planted in a line in wooden flats (24 x 18 x 5 inches) containing field soil mixed with farm yard manure. Each treatment was replicated four times in four different flats and nontreated seed was used as control. The experiment was repeated three times during the months of August and September. As far as possible, all seeds were planted at the same depth. The number of seedlings in each treatment were counted from the time emergence started and continued up to a week. Final counts were made 2 weeks after the date of planting.

RESULTS AND DISCUSSION

Results of the three experiments are presented in Table 1. All the fungicides with the exception of Leytosol increased germination to a considerable extent in comparison with non-

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The author is indebted to Dr. Dharampal Singh, Head of the Centre for valuable suggestions and help.
[2] Anonymous. 1957. Gibrel, Tech. Bull., Merck and Co.
[3] Anonymous. 1957. Gibberellic Acid, Tech. Bull., I. C. I.

treated seeds. There is some variation in effectiveness of various chemicals, and materials like Ceresan wet, Spergon, captan, Dithane Z-78, Panogen and Arasan have proved much superior to others. Leytosol proved toxic to the seeds and decreased the percentage of germination in all tests. It also produced various abnormalities in the radicle and plumule. All materials that improved germination also hastened emergence to a certain extent, and seedlings arising from treated seeds appeared more healthy and vigorous than the control.

Table 1. Effect of fungicides and gibberellic acid on germination of bajra (pearl millet).

Treatment	Percentage germination		
	Experiment 1	Experiment 2	Experiment 3
Ceresan dry	58	40.5	36.5
Ceresan wet	82.8	67.5	63.0
Agrosan GN	57.8	34.5	30.0
Leytosol	25.5	11.0	11.0
Leytosan	60.0	37.0	22.0
Spergon	89.1	53.5	38.5
Captan (Flit 406)	77.0	45.5	44.0
Dithane Z-78	77.5	52.5	44.0
Panogen	81.5	68.0	53.5
Arasan	80.5	67.5	54.5
Phygon	68.0	30.0	27.0
Gibberellic acid + captan	82.0	54.0	46.0
Gibberellic acid + Ceresan dry	63.0	43.0	33.5
Control	48.5	14.5	10.5

Incorporation of gibberellic acid into captan and Ceresan resulted in only a slight increase in percentage germination. This increase due to gibberellic acid does not seem to be significant. However, all seeds treated with hormone produced seedlings which were taller than those which resulted from seeds treated with fungicides alone or from non-treated seeds.

On the basis of these preliminary experiments, it can be concluded that treatment of pearl millet (bajra) seeds before planting is beneficial and can result in better crop stand. Whether these treatments can result in increased yield in the field has yet to be investigated. However, the use of fungicides will reduce the heavy seed rate generally used for this crop, and thus result in reduced cost of cultivation.

These results do not indicate that gibberellic acid can increase the efficiency of seed protectants. It seems plausible that mixing the hormone with the fungicide will result in healthier and better seedlings.

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NOTES ON HEMICRICONEMOIDES GADDI FROM CAMELLIAS IN LOUISIANA AND GEORGIA[1]

Leigh S. Whitlock and Arnold E. Steele[2]

In 1949 Loos (2) described a new species of Criconemoides from a tea-nursery soil in Ceylon and gave it the specific name gaddi. Chitwood and Birchfield (1) transferred this nematode to the genus Hemicriconemoides, which they established to embrace the species intermediate between Criconemoides and Hemicycliophora. Recently specimens of what appear to be H. gaddi were found feeding on camellia roots (Camellia japonica) in Louisiana and Georgia. The authors noted several deviations from the original description given by Loos and felt that their findings should be published. The specimens used for measurements and photographs were killed by gentle heat, placed in 5% formalin and 1% acetic acid, and then mounted and observed immediately (Table 1).

Table 1. A comparison of measurements of Hemicriconemoides gaddi found on camellia roots in Louisiana and Georgia with those found by Loos in tea-nursery soil in Ceylon.

Items measured	Loos (4 ♀♀)	Louisiana (10 ♀♀)	Georgia (10 ♀♀)
L	431-504 μ	570 (526-640) μ	536 (471-606) μ
a	15.8-18.0	16.4 (13.9-20.4)	15.4 (13.2-18.5)
b	3.7-4.4	4.2 (3.8-4.5)	4.1 (3.1-5.0)
c	19.9-21.7	17.7 (15.1-20.0)	16.8 (13.5-20.5)
V	91.2-92.3%	90.9 (89.7-92.1)%	90.7 (89.2-92.0)%
Stylet	72-80 μ	91.3 (85.2-98.0) μ	90.2 (84.9-97.7) μ
Annules	About 120	125 (119-128)	123 (115-130)

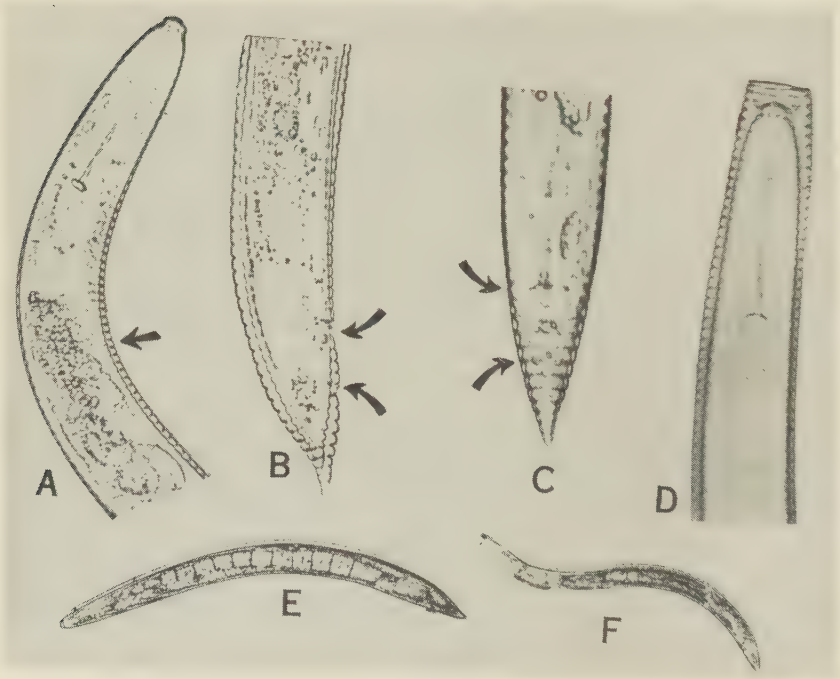

FIGURE 1. Hemicriconemoides gaddi ♀♀. A -- Anterior lateral view (arrow marks excretory pore). B -- Posterior lateral view (arrows mark vulva and anus). C -- Posterior ventral view (arrows mark vulva and anus). D -- Anterior view with sheath in forward position. E and F -- Full-length views with sheath in normal and shifted positions, respectively.

[1]Cooperative investigations of the Crops Research Division, Agricultural Research Service, United States Department of Agriculture, and the Georgia and Louisiana Agricultural Experiment Stations.

[2]Nematologists, Crops Research Division, Agricultural Research Service, United States Department of Agriculture, Baton Rouge, Louisiana and Salinas, California, respectively.

OBSERVATIONS

The head is recorded by Loos as not being offset, but in the Louisiana and Georgia specimens the head is definitely offset (Fig. 1 A, D). The sheath appears to be attached only at the base of the first annule (Fig. 1 A, D) and at the vulva and anus (Fig. 1 B). In a number of specimens, about 5 to 10%, the sheath has a tendency to slip forward and form a collar about the head (Fig. 1 D, F). Males were found in the Louisiana samples, but as specimens of Criconemoides and Criconema also were present, the true identity of these males could not be determined. Other measurements and descriptive points agree closely with those of Loos, except that the Louisiana and Georgia specimens are larger and have proportionately longer stylets.

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CROPS RESEARCH DIVISION, AGRICULTURAL RESEARCH SERVICE, UNITED STATES DEPARTMENT OF AGRICULTURE

PSEUDOMONAS SOLANACEARUM IN ISRAEL[1]

Zafrira Volcani and J. Palti[2]

The bacterial wilt caused by Pseudomonas solanacearum E. F. Sm. was first observed in Israel on potatoes in 1946 (1, 2). This note describes inoculation tests and observations of the conditions under which wilt outbreaks have since occurred in this country.

HOST RANGE AND STRAIN OF PATHOGEN

Field occurrence of the wilt has so far been limited mainly to the potato crop, and has been observed only on rare occasions on tomato plants in the Hula Valley. However, in artificial inoculations tomato plants have been found to be very sensitive to the disease. A careful survey failed to reveal any sign of wilt infection on groundnuts -- although this crop frequently succeeded wilt-infected potato crops -- nor under natural conditions on any of the other hosts mentioned in literature (2), such as tobacco, eggplant, Chili pepper and banana.

Host range studies by the senior author in cooperation with Dr. F. S. Littauer and Mrs. N. Temkin-Gorodisky of the Division of Storage Research of Fruits and Vegetables, have shown that only eggplants can be infected by the pathogen under certain conditions, while tobacco, groundnuts and Chili pepper were not affected. Infection tests were made in four ways: (a) sowing or planting in soil from which a severely affected potato crop had previously been lifted; (b) sowing or planting in healthy soil infested with diseased potato tubers; (c) dipping roots of young tomato, tobacco, eggplant and pepper plants in a water suspension of a freshly isolated culture of the organism, and subsequent planting in healthy soil; (d) growing plants in healthy soil, pricking them with a needle at the lower or upper part of their stem through a drop of water suspension of the pathogen, and covering them with bell-jars for 24 hours.

Of all the plants tested only potato and tomato were severely infected. Progress of infection was more rapid with plants inoculated by puncturing the stem or by dipping in a water suspension. Plants showed wilting symptoms 5 to 10 days after inoculation. Eggplants, on the other hand, were infected only when pricked at the stem. Young plants were far more sensitive to the disease than older ones.

Parallel infection experiments made on the same hosts with a culture of P. solanacearum, sent by Dr. A. Kelman[3] from North Carolina, caused wilting of potato, tomato, tobacco, pepper and eggplant. The Israeli isolates differed from the American organism not only in their pathological host range but also in some of their morphological and biochemical traits, and are to be considered a different strain. It is interesting to note that year after year the same strain has always been isolated from infected plants, wherever the disease was found in Israel.

FIELD SURVEY

Distribution: Bacterial wilt occurs in Israel both on potato crops sown in spring and lifted in June-July and on crops sown in August and lifted in November-December. The losses caused by the wilt are heavier in the spring than in the autumn crop.

The disease is endemic in only one part of Israel, the Hula Valley, where light to severe infections of potatoes are found almost every year. In all other parts of Israel appearance of the wilt is sporadic and rare.

Soils: The wilt disease appears on a wide range of soils. In the Hula Valley it is found on calcareous soils as well as on medium loams and heavy clay soils. In other parts of Israel the disease has been recorded on all these types of soil and on loess soil.

Host Age: Although in the inoculation tests young potato plants appeared very sensitive, we have never observed the wilt in young potato crops. The lowest age at which the disease appeared was 50 to 55 days after sowing. Rapid progress is made by the disease where the crop matures under high temperature conditions, which explains the severe infection of spring crops. In autumn crops, on the other hand, maturity occurs under lower temperature conditions and wilt incidence is restricted.

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[1]Publication of the Agricultural Research Station, Beit Dagan-Rehovot. 1960 Series, No. 325-E.

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Control: Control consists principally in securing disease-free seed tubers. This presents no difficulty in Israel, as the disease is rare outside the Hula Valley.

In the infected Hula soils losses from wilt can be reduced by sowing the spring crop earlier and the autumn crop somewhat later than is the usual practice. But earlier spring sowing is possible only if there is a suitable interval between rains in February. Late autumn sowings run the risk of encountering the December rains which may interfere with lifting.

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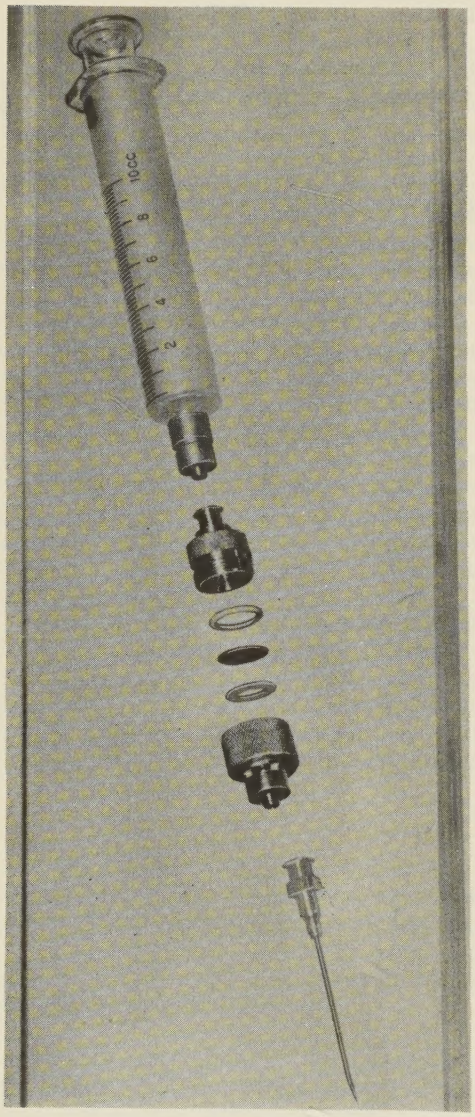
AGRICULTURAL RESEARCH STATION, BEIT DAGAN-REHOVOT, ISRAEL

A MODIFICATION OF THE BÜCHNER FUNNEL METHOD FOR TRANSFERRING AND CONCENTRATING NEMATODES[1]

Leonard W. Storm[2], Nancy S. Storm[3], and Donald A. Dahlgren[2]

In the course of recent work involving anatomical studies of the larval form of the citrus nematode, Tylenchulus semipenetrans, it became apparent that a rapid method of concentrating and transferring these organisms was needed.

After the nematodes were extracted from the soil, by Christie and Perry's technique[4], the extracted solution containing the citrus nematode was taken up in a Luer-Lok hypodermic syringe. A swinny hypodermic adapter (Millipore Filter Corporation Catalog XX 30 01200) containing a Millipore H. A. filter (Fig. 1) was attached to the syringe and the liquid was slowly expressed from the syringe. The filter was then removed and the nematodes were eluted off the filter in the killing solution. This procedure was repeated as the nematodes were taken through each of the washing and alcohol dehydration solutions. In principle this method is similar to the Büchner funnel technique, reported by Feder and Feldmesser[5], but it is more rapid and convenient to use.



(FIGURE 1. (Courtesy of the Millipore Filter Corporation).

Later experiments showed that large quantities of material extracted from soil by Christie and Perry's technique could be concentrated to a few milliliters in a very few minutes. After the nematodes were eluted off the filter they were checked by a dissecting microscope and found to be alive and apparently unharmed by the treatment.

This method should have wide use when there is need for concentrating nematodes for inoculation experiments or for anatomical studies, where it is necessary to transfer nematodes from one solution to another. It cannot be used when the solution involved is methacrylate, as the filters are dissolved in this material; however, in all other instances many hours can be saved by this technique.

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[1]Contribution from the Arizona Agricultural Experiment Station. Journal Paper No. 576.
[2]Research Assistants, Department of Plant Pathology, University of Arizona.
[3]Research Assistant, Department of Agricultural Biochemistry, University of Arizona.
[4]Christie, J. R., and V. G. Perry. 1951. Removing nematodes from soil. Proc. Helminthol. Soc. Wash. D.C. 18:106-108.
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BOOK REVIEW

"THE PATHOGEN." Volume II of the three-volume advanced treatise on Plant Pathology, Edited by J. G. Horsfall and A. E. Dimond. Published by Academic Press, New York-London. xiv + 715 pages. 1960. Price $22.

"The Pathogen" measures up in every way to the high standards established by Editors Horsfall and Dimond in their first volume, "The Diseased Plant." Volume III -- "The Diseased Population" -- which is scheduled for issue later this year, will complete the set. We would anticipate that the three books would enable active professionals not only to become more knowledgeable, but more effective, whether they are engaged in teaching, research, extension, or industry.

Like the first, this volume includes contributions from several of the world's leading biological scientists, carefully organized and edited to advance the pathogen theme. And here again, the editors have, in their first chapter, publicly come to grips with what must have been their most difficult problem -- precisely defining and delimiting the contents of the volume.

Their approach to this problem has been a bold one: to give concise meanings to words basic to this second volume -- "pathogen", "parasite", "cause." These, of course, are everyday words to plant pathologists, but, as the editors make clear, they do not necessarily mean the same thing to each pathologist.

Horsfall and Dimond go back to antiquity to define pathogen as "an agency that gives birth to, or generates suffering" and urge, with good argument, that the word be used broadly, to cover all agencies that qualify. They include: (1) the animate pathogens -- microbes, nematodes, arachnids, and insects; (2) virus pathogens, and (3) inanimate pathogens such as nutritional deficiencies that cause diseases.

They note the rather unscientific approaches taken through the years in naming plant diseases; they are named from man's earliest history, from symptoms, the host's name, for the pathogens, and in some cases, for their discoverer. The authors suggest an improvement: define diseases in terms of the pathogen, using the suffix "al" meaning "to be characterized by." Thus, we would have fungal diseases, bacterial diseases, viral diseases, fusarial diseases, phytophthoral diseases.

The editors also strongly and effectively defend "cause" over the newer word "incite" in describing how diseases result.

The 15 chapters that make up volume II are concerned not only with a broad examination of pathogens, but with consideration of such specific phenomena as the mechanical and chemical abilities of pathogens to breach the host barrier; the interaction of pathogen, soil, and host; toxins; heterokaryosis, saltation, and adaptation; fungicidal chemistry, and nematocides.

The list of foreign scientists contributing to "The Pathogen" includes: from England, F. C. Bawden and E. W. Buxton, Rothamsted Experimental Station; Sydney Dickinson, University of Cambridge; Lilian E. Hawker, University of Bristol; and R. K. S. Wood, University of London; from Canada, T. Johnson and R. A. Ludwig of the Department of Agriculture; from India's University at Madras, T. S. Sadasivan and C. V. Subramanian; and from New Zealand, R. E. F. Matthews of the Department of Scientific and Industrial Research.

Contributors from the United States are the editors and Saul Rich, all from the Connecticut Agricultural Experiment Station; M. W. Allen, University of California; Vincent W. Cochrane, Wesleyan University, Middletown, Conn.; Carroll E. Cox and Hugh D. Sisler, University of Maryland; and George L. McNew of the Boyce-Thompson Institute. -- PAUL R. MILLER

RESULTS OF 1959 FUNGICIDE AND NEMATOCIDE TESTS

The "Results of 1959 Fungicide and Nematocide Tests" is now available. This report is issued annually by the American Phytopathological Society Advisory Committee on Collecting and Disseminating New Fungicide Data. This report serves as a medium for organizing and presenting the summarized results of current fungicide and nematocide testing projects. Much of the information is never otherwise published or made conveniently available. Information on products available for testing, composition of products and their sources are given.

Copies of this report are available at $1.00 per copy when accompanied by a remittance, $1.25 when invoiced and billed. Address orders to A. B. Groves, Winchester Fruit Research Laboratory, Route 3, Winchester, Virginia. Make remittances payable to the American Phytopathological Society.